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THE IMPACT OF PROTOZOA ON WORLD ECONOMIC PROBLEMS*

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In the short time at my disposal it will obviously be impossible for me to do more than skate around the fringes of my subject, but I hope to produce enough evidence about some of the more important protozoa affecting the economy of mankind to indicate beyond any cavil or question that the subject of my address deserves immediate, energetic, and well-directed action.

The phylum Protozoa is a large one both in number of species and in distribution on the earth and in the water, and its members exercise an influence in these spheres which, while immense and essential, is still merely one part of the integrated ecology participated in by animals, plants, and inanimate nature. The part played by protozoa in this ecological complex is too vast a field to be more than mentioned, and so I propose to concern myself only with some of the parasitic protozoa which, by their direct action, affect the social economy of man. As examples of these I shall confine myself to three groups of protozoa which I have chosen as examples partly because of the great importance they possess for man, but also because they are groups upon which man can exert a direct influence to lessen their impact on his economy. The three groups I have chosen are the trypanosomes, the plasmodia, and the piroplasms.

THE TRYPANOSOMES

By this term I do not wish to imply the whole family Trypanosomidae, but merely the genus *Trypanosoma*. The trypanosomes have been recognized as of importance to the economy of man since the discovery of Evans in 1880 of a trypanosome which caused "surra," a disease of horses, camels, and other animals. In 1902 Forde was the first to recognize trypanosomes as a cause of disease in man in Africa, and the knowledge which has since accumulated of their ubiquity in and importance to the domestic animals of man, especially in the African continent, has made them one of the major problems requiring attention if meat and dairy products, which constitute so important a part of man's dietetic needs, are to be made available in quantities adequate for the continually growing population of the world.

I propose here to do no more than mention the ravages produced by trypanosomes in man on the east and west coasts of Africa and in South America, important as the effects of these were and are on the distribution, vitality, and general welfare of the populations affected. This is because, from the broader aspects of man's economy, I consider the trypanosomes affecting man's domestic animals to be far and away the more important when the whole picture is looked at in perspective. As Hornby (1949) has stated, trypanosomiasis has the unique distinction of being the only disease which *by itself* has denied vast areas of land in Africa to all domestic animals except poultry. These areas total one-fourth of the land surface of the continent. Apart from these completely unstocked areas, there is a still greater aggregate of land surface in every continent except Australia where trypanosomiasis is one of the major scourges of stock. The factors concerned and the results brought about by trypanosomiasis can be illustrated by Vogt's formula $C = B:E$, where C is the carrying capacity of the land, B is the biotic potential, and E is the environmental resistance. In this study I wish to indicate how much C is lowered by the part of E due to trypanosomiasis.

Still limited by my time allowance, I can consider only bovines among man's domestic animals, but, as these are among the most important of animals used by him as food, the effect of infections on them will serve as a measure of the full effect of trypanosomes in reducing the quantity and quality of beef supplies and dairy products.

Three Main Groups

The most important trypanosomes pathogenic to bovines fall into three main groups, which, in the absence of easily applied differentiating specific characters, I shall refer to as the *vivax* group, the *congolense* group, and the *brucei* group. In some areas one is the prevalent pest, in others another, while very often two or even all three may coexist and take their toll of stock. The members of all these groups are carried from animal to animal by various species of tsetse fly, so that the protozoological problem now becomes linked with an entomological one. While cyclical transmission by tsetses is the normal method, it would be unwise to assume that direct mechanical transmission by other biting flies is of little importance. The proof of this is the establishment of foci of *Trypanosoma vivax* endemicity in South and Central America, Mauritius, and the West Indies, where tsetses do not now occur.

The distinction between these three groups is relatively simple from the entomological angle, as the dissection of infected flies will supply the answer in any given area. When tsetses display the presence of cyclical forms in the salivary glands we can say the

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infecting trypanosome belongs to the *brucei* group. When the infection is found both in the midgut and in the mouth parts, the cyclical forms being in the latter situation, the trypanosome belongs to the *congolense* group, while the presence of multiplying trypanosomes in the mouth parts alone indicates the *vivax* group.

It would be fruitless to make any estimate of the bovine population of even one continent such as Africa, and much less of the world. Of all the continents, however, it is in Africa that trypanosomiasis ranks most highly as a cause of cattle losses, and it would be interesting, therefore, to know the degree of risk to which cattle are exposed in, or passing through, fly belts in Africa. Mere impressions are often unreliable, and the amount of accurate evidence on this point is limited, but certain detailed observations recently made by Unsworth (1951) are revealing.

Mortality and Morbidity Due to Trypanosomiasis

In the course of experiments devised for other purposes, 10 control cattle were introduced into an area infested with *Glossina palpalis*. All contracted infection with trypanosomes and all died within 146 days. The trypanosome concerned was *T. vivax*.

In similar experiments in another area 10 control cattle were introduced into an area infested with *Glossina morsitans*. All became infected with trypanosomes, eight died within 81 days, one died after 154 days, and one escaped after 110 days but was later found dead—probably owing to trypanosomiasis. The trypanosome concerned was again *T. vivax*. Admittedly these figures are small, but they relate to carefully controlled experiments and are an indication of the losses which would be sustained by unprotected cattle in areas infested by either *G. palpalis* or *G. morsitans*.

These experiments, while striking evidence of the potentially disastrous consequences of bringing cattle into such areas, need not be regarded as necessarily applicable everywhere. There is good evidence to show that there are considerable strain variations in virulence, so that *T. vivax* in one area may produce a virulent infection with high mortality, while in another area the infection may be mild with low mortality.

Mortality, however, is not the only criterion of the damage sustained by herds due to trypanosomiasis. Affected cattle, even if they do not die, often lose condition to a marked degree, are emaciated and weak, and such animals, if trekked to markets or otherwise subjected to strain of any kind, will often succumb when they might have survived if rested. But rest is not the fate of most cattle in Africa. The cattle of the Fulani, those nomadic herdsmen, have to follow their food, and, as the seasons of the year change and grazing grounds alter, must accommodate their movements accordingly. These wanderings sooner or later bring them through fly belts, and the consequences, in the absence of protective measures, are inevitable. The only possible way to prevent this wandering habit, which, after all, is dictated by the need for water, would be the provision of permanent water supplies, but, as this is a matter for engineers and is outside the scope of my paper, we need not pursue the subject further, although it will come in for brief mention at a later stage.

Mortality and loss of condition, however, are only part of the failure to reach maximum productiveness in numbers and economic usefulness of bovine resources. These are direct losses in existing herds, but quite immeasurable are the indirect losses due to the exclusion, on account of the presence of trypanosomes and tsetses, of what would be vast potential herds from pastures otherwise admirably suited for them. Still more indirectly one has to consider the losses due to overstocking in fly-free areas on account of the restriction of grazing grounds due to the presence of tsetses. Such losses include also the effects of loss of condition, helminthic and bacterial diseases, and infertility.

All this means that the mere mortality in herds due to trypanosomes in any particular area is only a very partial picture of the losses, actual and potential.

Control Measures

This brings us, logically, to consider control measures. These, although outside my present subject, must be touched upon, as they are the natural concern of all who are faced with the practical control of trypanosomiasis. They resolve themselves into measures, direct and indirect, against the tsetse and direct measures against the trypanosome, or a combination of these.

Direct measures against the tsetse are confined to the use of insecticides. Such means may be of value against species such as *G. palpalis* with a limited range or a range limited by environmental factors, such as belts along river courses, but are at present inapplicable or would be inordinately expensive against species such as *G. morsitans*, widely dispersed over large areas. In any case, the indiscriminate use of insecticides on such a large scale might have unexpected and harmful effects due to unselective slaughter of insects.

Indirect measures have probably a wider and more useful application. These consist of selective clearance of bush under skilled supervision in the case of riverine tsetse, and settlement and agriculture of fly-infested areas, where this is possible, in the case of the more widely dispersed species, such as *G. morsitans*.

I do not propose to go into the subject of bush clearance, which is a highly specialized affair, but would merely draw attention to my use of the word "selective" in regard to bush clearance. Preservation and improvement of water supplies is of paramount importance in Africa, and therefore indiscriminate removal of blocks of forest or of fringes of forest along river courses is indefensible if it can be avoided. Possibly, chemical means of destruction of tsetses along the edges of sharply cut corridors in the forest would suffice, the corridor giving access to the river for watering.

G. morsitans is essentially a "game" fly, and goes when game goes. In this way agricultural development and settlement alone will remove it. As food, it prefers game to cattle and cattle to man, but it must be remembered that mere clearance of game without alteration of the other conditions suitable for *G. morsitans* might merely substitute cattle as a perfect alternative to game. On the other hand, agricultural development means an increase in the human population, with consequent and essential changes in the fly's environment, until an increase in the population up to 200 or over per square mile will so alter conditions as to eliminate *G. morsitans*.

We have here been considering only the control of tsetses, but it must not be forgotten that mechanical transmission of trypanosomes by biting flies may be an important method of transmission. *Trypanosoma evansi*

is transmitted wholly by this means, although there is a distinct possibility that it may once have been cyclically transmitted by tsetses but have lost this capacity, while *T. vivax* has spread far beyond tsetse areas and maintained itself by mechanical transmission, still retaining its transmissibility by tsetses. Thus, as already mentioned, it is now firmly established in South America and other tsetse-free areas.

Prophylaxis

Direct measures against the trypanosome are treatment of infected animals and prophylactic treatment of cattle. There are drugs now available, and more seem to be on the way, which are effective in the treatment of trypanosomiasis in cattle. This efficacy, however, is not equally potent against all species of the trypanosome. One of the latest of these drugs is phenanthridine 1553, which has given striking results from treatment by a single subcutaneous injection of 1.5 mg. per kg. body weight of a 1% solution. Cases of photosensitization following this treatment have been remarkably few. Successful as this treatment has proved, such drugs may be of still greater value when used prophylactically on herds which have to pass through fly belts or have to live in fly-infested areas. Such use would not only prevent infection of the cattle but would protect the cattle of uninfected areas from infection by imported infected cattle.

The value of "antrycide" used as a prophylactic has been investigated in a series of carefully controlled experiments by Unsworth (1951), whose conclusions were:

> "It would appear that the effectiveness of antrycide prophylactic mixture in protecting cattle against infection and death from trypanosomiasis depends very largely on the intensity of the trypanosome challenge to which the cattle are exposed. Where the quantum of infection is low, such as might result from exposure to riverine tsetse in Northern Nigeria, the inoculation of cattle at three-monthly intervals with antrycide prophylactic mixture will give complete protection against death and will protect 88% to 95% of cattle against infection with trypanosomiasis. The small proportion of cattle in which trypanosomes do 'break through' the drug are apparently cured by subsequent inoculations. Where, on the other hand, the quantum of infection is great, such as might result from exposure to an average challenge from *G. morsitans*, the inoculation of cattle at three-monthly intervals with antrycide prophylactic mixture cannot be relied upon to give complete protection, either against infection or against death from trypanosomiasis. Moreover, such a procedure is not unattended by danger, for in the experiment which we have just described at Lumu there were indications of the development of an antrycide-resistant strain of trypanosome, eight months after the cattle had first been introduced to the area."

His conclusions indicate the uses and limitations of antrycide as a prophylactic, and may be taken to summarize the present position as regards prophylaxis.

A Controversial Question

Having now considered the effects of trypanosomes on human economy we have to deal with the more controversial question of what might happen were we able by one means or another altogether to eliminate the pathogenic cattle trypanosomes.

I must refer again to Vogt's formula C = B:E. C, representing the carrying capacity of an area, would not necessarily be augmented by eliminating trypanosomiasis. Even if we leave out of consideration other bovine diseases, protozoal or otherwise, there remains man's cupidity, which might result in his attempting to keep a larger stock of animals than the land at his disposal could with advantage support. It may be, therefore, that trypanosomiasis acts as one of the checks on this evil, and that careful thought should be taken before removing it entirely in any care-free manner.

We have to bear in mind the fact that although tsetses limit the areas where stock can thrive or even exist, yet they are not a bar to their existence to the extent that many people imagine. On the contrary, there are great areas where large numbers of domestic animals succeed in existing in contact with tsetses so long as the latter are not numerous or effective enough to cause repeated infections.

In areas from which the presence of tsetses utterly debars stock, though otherwise suitable for them, the removal or reduction in numbers of tsetses would allow the land to be made use of and so greatly increase the carrying capacity of the country and lead to a better distribution of stock. This would also tend to prevent overstocking of areas at present usable and overstocked. Tsetses, however, are not the only factor influencing distribution of stock; there is, as I have already said, the distribution of permanent water, which in its turn influences the distribution of human population, with consequent effects on agriculture and the diminution in numbers of tsetses which follows this sequence. There is a third factor governing distribution of stock which we consider in another place—namely, tick-borne disease; and yet a fourth, helminthic disease, which, however, is outside the scope of this paper.

Were we to remove, if it were possible, all these factors limiting the increase of stock, the numbers of the latter would certainly lead to overstocking and greatly accelerated soil erosion, so that there would result an economic evil which would be largely irreversible. This same overstocking would also result in an increase in the incidence of many other diseases of stock—viral, bacterial, protozoal, and helminthic—so that better distribution would be of all-round benefit. However, the complete removal of limiting factors is not in sight, and I only sound a warning. On the other hand, there is no doubt that tsetses do cause overstocking of areas free or relatively free from flies and so hasten soil erosion in them. The removal, therefore, of tsetses or of trypanosomes would allow a better distribution of stock and so act indirectly as a measure of soil conservation. It would also help in more widespread use of composting to fertilize the hungry soil.

To sum up, the menace of trypanosomiasis is bound up with many other menaces, and all these should be considered as an intimate complex in devising and putting into effect measures of limitation or prevention. Only in this way can we ensure that man's economy in regard to more and better supplies of meat, milk, dairy products, and agricultural crops shall be best served and debilitating deficiency diseases be reduced.

THE PLASMODIA

In the previous section it will have been seen that the impact of protozoa on man's economy was primarily on his food supplies, and thus only indirectly on man himself. In the case of the plasmodia the direct impact is on man and only indirectly on his food supplies and general economy. The impact in this case has a much

wider contact than in the case of the trypanosomes, for there is no continent where plasmodia do not exert a major influence on man's activities, even to the extent, in some tropical areas, of prohibiting them entirely.

Now when one considers the relatively low mortality from malaria over the entire population, even in areas of high endemicity, one might imagine at first that the effect on man's general economy would be small, whereas we know that, in truth, the effect is enormous. This apparent paradox is due to the fact that the influence of malaria is exerted for the most part indirectly. While relatively few die, enormous numbers are debilitated to a greater or lesser degree, work hours are lost, physical capacity for work is lowered, mental capacity is impaired, and the vigour and vitality of a people are diminished. All these damaging results are roughly proportional to the degree of endemicity, and I am in disagreement with those who assert that a very high degree of endemicity has little effect on the economy of the people affected. The argument of this school of thought appears to be that in such an area there is a high degree of immunity, or rather tolerance, in the adult population and the adult is looked upon as the main or chief economic unit. This seems to me to be a very one-sided way of looking upon the picture.

In a community such as that envisaged, the high adult tolerance is accompanied by a high infant mortality, and in assessing the effect on the general economy of the community in a long-term view this wastage must be taken into account. Among figures given by Wilson (1936) for a tribe living in Tanganyika in an area such as we are considering, the average number of children surviving to women who had completed their child-bearing life was only 1.13. If we take the sex ratio as approximately 1 : 1, this means that only one woman in two would have a surviving female child to produce children of her own. It is only too evident that such a community would be a dying one unless reinforced by immigration.

It seems to me quite irrational to maintain that such a community has a healthy economy because its adult population shows a tolerance to malaria, enabling the adult unit to work more or less continuously and with a minimum of lost working hours. It may be true that the working units are in a stable condition of health as regards malaria, but the community as a whole is like a man living on his capital and having no regularly paid income to keep him in a financially sound condition.

In order to get some idea of the total effects of the plasmodia on man's economy one has to consider this economy as a many-sided figure and to examine each facet separately.

In pre-human days, when man was a higher ape wandering in the forests in smaller or larger family groups, it is possible that he lived, as some of the primates now appear to do, in a partnership with his malaria parasites. Each group would occupy a relatively limited tract of country, and the aggregate of all these groups would be infinitely small as compared with present human populations. Each group, which might be composed of several families, would be infected with its own strain of malaria parasite, and in the course of time would develop an immunity or at least tolerance to that strain on evolutionary lines by the continued survival of the more resistant individuals. A time would arrive when the prehuman being and his malaria parasites would have come to a *modus vivendi* whereby mammal would provide a habitat to protozoan, and protozoan would cause a minimum of inconvenience to mammal.

The prehuman being, however, having a divine discontent with his limited surroundings, began to wander outside his group's natural terrain and so came in contact with other groups. In this way adjacent groups might amalgamate into larger communities, which probably accelerated the human evolutionary process and led towards much greater agglomerations of fully human communities. This led inevitably to man coming into contact with strains of plasmodia foreign to his old economy and against which he had little or no immunity. In other words, part of the price man paid for progress towards true manhood was an increasing tribute to malaria.

We may now consider the forms which this tribute took and the effect it had on man's economy.

Effects on Distribution of Population

When one examines the distribution of people on maps of the world it becomes evident that the most important single factor encouraging or limiting this distribution is the presence or absence of permanent surface fresh water, in the forms of rivers or lakes.

It does not follow from this that areas with the most lavish water supplies are necessarily the most thickly populated, because other factors besides water are in operation. This would apply, for instance, in the great Amazon basin, with its mighty rivers and forests but very sparse human population. On the other hand, scarcity of water is always accompanied by a sparse population, as in the desert or semi-desert areas. That this sparsity is due to lack of surface water is exemplified by the relatively small population in most of Iraq and the country between Mosul and the lower Tigris and Euphrates, where formerly a much denser population with thriving cities, such as Ur, Babylon, Nineveh, and others, once existed owing to a vast irrigation system of communicating canals. When these fell into disrepair owing to various causes, the ravages of the Mongols among others, the cities and the thriving population were swallowed up in the arid desert. It would appear, therefore, that on a world scale malaria does not play a decisive part in the distribution of population.

Plenitude of water, however, facilitates occupation by both mosquito and man, the latter on account of its use for his agricultural activities. According to Macdonald (1950), in some such rich agricultural areas in Africa *Anopheles gambiae* has been unable to prevent a concentration of population, although in India and Ceylon there are large areas where, in the absence of preventive measures, *A. fluviatilis*, *A. minimus*, or *A. culicifacies* deny the use of the land for human settlement. Thus in certain foothill areas of the Himalayas and of the Western Ghats of India the country is almost uninhabited owing to the presence of the almost exclusively anthropophilic *A. fluviatilis*.

Effects of Redistribution of Population

We have so far been considering the effect of malaria on more or less static populations, but we must now look into what happens when population movements on a greater or lesser scale take place. The causes of

such displacements are varied. Migration of population may be due to the pressure of overpopulation in healthy and prosperous areas, causing a spill over into more malarious country. It may be due to mass movement of labour to areas where public works such as dams or bridges are being constructed, or of labour required in mining and similar large-scale operations. There is a third form of two-way migration in certain parts of India, where a population normally engaged in agriculture flocks in large numbers to cities, such as Bombay, to work in cotton mills during the off season in agriculture and returns to the latter occupation for the sowing and harvesting seasons.

In all these cases, except possibly the last, the tendency is for the population to move from a less malarious to a more malarious area, but this is not invariably the case. The effects, however, in the absence of preventive measures are the same—an increase of malaria sometimes on a disastrous scale, either on the immigrants or on the population of the area invaded. Although many other factors come into operation in producing malaria in epidemic form under the conditions cited, the most important factor is probably one inherent in the malaria parasite itself.

It is well known, and can easily be experimentally demonstrated, that the immunity to malaria parasites is species-specific. This means that a mammal infected with one species of malaria parasite cannot be superinfected with the same species, but may easily be infected with any other species to which it is susceptible. In other words, a man carrying *P. vivax* cannot be superinfected with *P. vivax* but can easily be infected with *P. falciparum* or *P. malariae*. This specific premunition goes even more deeply than species, because it is found to be not merely species-specific but also strain-specific. Thus a man carrying a strain of *P. vivax* acquired in area A cannot be superinfected with that strain, but can be superinfected with a strain of *P. vivax* derived from area B. These facts supply the clue to the danger of introducing populations from non-malarious to malarious areas or even of introducing populations well adapted to one species or strain of parasite to an area where a different species or strain is present.

Where no precautions are taken such movements of population may have disastrous results. To cite a case coming within my own experience, I may mention what happened in one area of Assam during the recent world war. In the mainly rice-growing district of Sylhet the country is very low-lying, a mere 20 ft. or so above mean sea level, although situated 150 miles from the sea. The communications are by earth roads raised above the level of the surrounding country. The villages are mostly situated on the highest ground available. During the monsoon the whole country is inundated and the villages exist as island areas connected by the raised roads. In spite of these conditions the district was relatively non-malarious and the population non-immune. Large numbers of the men had gone to work as labourers on military works situated in malarious districts. When these returned to the area in question many were infected with strains of malaria parasites from the districts in which they had been working. The result was disastrous. A suitable carrier species of anopheles already existed; out of a population of about 40,000 people in this particular area some 7,000 to 8,000 died of malaria before any effective action was taken.

This is an instance of an infected population moving into an area of very low endemicity, but the results may be equally unfortunate when a comparatively healthy population enters an area of high endemicity or, as already stated, even when a so-called "salted" population moves into an area where their immunity to their own strain of malaria is comparatively inoperative against the new strains or against a different species of parasite. In other words, almost any kind of redistribution of population in malarious areas, even when the endemicity is not high, is likely to be accompanied by severe epidemics of malaria.

The possible economic effects of such disastrous epidemics will be only too obvious. The life of the community is completely dislocated, and this may be accompanied by a state of apathy that prevents any energetic action by the community itself, which can be saved only by outside help.

Effects on the General Life of a Community

This is a very comprehensive aspect, and includes effects on agriculture, industry, social services, including education, and the general welfare of the population.

Looked upon from this angle, the effect of malaria on a community may mean anything from a slight inconvenience to an almost complete cessation of individual and community activities. In the milder degrees of malarial endemicity the chief effect will be in the form of days lost to all beneficial activities on account of illness among the workers. This effect may be much greater than would be expected from the number of days lost, especially in tropical communities where, as Macdonald (1950) has pointed out, the success or otherwise of agricultural operations very often depends on the climatic conditions obtaining during a limited part of the year, generally that part when ploughing and sowing are carried out. If the sickness among the population coincides with this period the days lost and the impaired physical condition of the workers will have an effect on their productive capacity the results of which will endure throughout the remainder of the year in a way which would not have been the case had the malaria been prevalent at a less important part of the annual work cycle.

The effect will be further enhanced by the fact that children as well as adults are sure to be affected, and in most primitive communities the older children are themselves economic units in the community.

This represents the effect on the economy of a community working for itself, but under the same conditions the effect is the same when such a community is employed as labour in some local industry. The absenteeism and the diminished physical capacity, added to wastage of labour, needing new recruitment, and to provision of special measures of housing and medical care, will inevitably lead to loss of time in completion of projects and to a greatly added cost. There is no aspect of the life of the community which escapes the impact of malaria even in the milder forms we are considering. The future economic units, the children, are frequently ill, leading to absenteeism, which interferes with education and may in many produce a constantly recurring invalidism, conditions under which the child will certainly not receive the full benefits of attendance at school, and the money and other resources

applied to an educational policy will not bring commensurate results until the harmful influence of malaria is eliminated.

To proceed from a consideration of the conditions we have been contemplating where malaria merely slows down the pulse of a community, let us see what happens when malaria in severe epidemic form lays a community prostrate.

Only if such a catastrophe has been personally seen can any conception be formed of the extent of the blow struck upon the community. Such epidemics usually occur in areas where malaria transmission is chiefly confined to certain periods of the year and seldom, if ever, in hyperendemic areas where transmission is going on throughout the year. Typical of such epidemics was what has happened in the Punjab on various occasions, in Ceylon, and in various other parts of the world.

During one of the Punjab epidemics a village in the affected area at the height of the epidemic presented an astonishing sight *and sound*. The following account was given me by an eye-witness:

> Most of the dispensaries of the district were closed because the staff were ill, and those open were neglected for the same reason. The countryside looked empty; cattle were wandering about unattended. As one approached a village a curious moaning sound was heard. This was the lowing of cattle waiting in vain to be milked. More than half the population were lying on "charpoys" unable to get up and neglected by all. Others were sitting about unable to work. Many huts had collapsed in the rain and remained unrepaired. Everyone looked half-starved, because none were well enough to grind the corn or fetch water for making "chappatis." Many young children and the old died of starvation, the former because their mothers could not attend to them. Many dead were lying untended on "charpoys" or on the ground. The village had ceased to exist as a human community. All the destruction was not directly due to malaria. Malaria was the *fons et origo mali*, but in its train starvation and dysentery played their part. Family ties were loosed, and brother did not care for brother or mother for child.

In case some of you should think this picture exaggerated, let me say that I have myself seen conditions where corpses were left unattended not only on the roads and by the wayside but even, in more populated places, on railway platforms. In other words, the whole organized structure of a community disintegrates on the failure of its human elements.

The general account I have given will leave no one in doubt about the profound effect of malaria on a community. The full effect may sometimes not be evident from a mere perusal of statistics showing the number of malaria cases occurring in a district and of mortality from malaria. A very striking instance of this was brought to my notice by Professor Macdonald. In Ceylon the total mortality over a period of nearly 50 years remained at a high relatively uniform level. In late 1946 effective antimalarial measures were begun, and in the next three years there was a very notable decrease in the *total* mortality. As the only notable new public health measure taken during this period was malaria control, the improvement can only be attributed to this, and is an indication of the direct, if not always apparent, effect which malaria exerts over the economy of a district, an effect which is much greater than would be expected from the known mortality due to malaria itself.

Sinton (1935) has calculated that in India alone 1,000,000 deaths a year are caused directly by malaria, another 1,000,000 are caused indirectly, and the number of cases of malaria occurring annually is 100,000,000. These figures seem incredible from one country alone, but on my putting this question to Professor Macdonald he said that, although at one time he was disposed to discredit the figures, his own subsequent studies have led him to the opinion that they may be an underestimate.

THE PIROPLASMS

The two types of protozoal parasites I have already dealt with, showing how severe is their impact on man's economy, are probably more familiar to most of you than the one I am now going to deal with—namely, a piroplasm, using this term in a broad sense. This is because both the former attack man as well as animal and so force themselves more directly upon our notice. The piroplasms, on the other hand, so far as we know, are animal parasites only, but their impact upon the animal food supplies of man makes them at least as important as the trypanosomes and plasmodia.

In the sub-order Piroplasmidea there are two families, the Babesiidae and the Theileriidae.

The piroplasms are parasites of a very large range of mammals—cattle, sheep, goat, pigs, horses, dogs, monkeys, antelopes, rodents, and other groups, so that their impact on mammals is a wide one. But, strangely enough, man seems to be immune, and the effect on his economy is entirely due to losses caused by this group to his domestic animals. I have already indicated the wide range of animals affected, but, again limited by my time allowance, I shall take as an example one group alone—the bovines. Cattle are parasitized by members of both the families I have mentioned, Babesiidae and Theileriidae, and each is the cause of various diseases of cattle, such as redwater, East Coast fever, and other diseases due to miscellaneous "small piroplasms." By far the most important of these diseases of bovines is East Coast fever, caused by the piroplasm *Theileria parva*, and I shall deal mainly with this in order to give a somewhat fuller account of its importance than could be attempted were I to deal with the other piroplasms as well.

East Coast Fever

In order to give an intelligible account of the effects of East Coast fever, I must describe, in the shortest manner possible, the life history of the causative parasite, as on a knowledge of this will depend methods of combating its menace to the food supplies of man.

East Coast fever is a blood-and-tissue infection of bovines with the parasites above mentioned, and is transmitted by various ticks such as *Rhipicephalus appendiculatus* and other species. The parasite in the blood is a minute organism 1–2 μ in diameter which attacks the red blood cells. Inoculation of blood into susceptible animals does not reproduce the disease, so presumably the bloods forms are gametocytes destined for sexual reproduction in the vector tick. Asexual multiplication takes place in endothelial cells of the internal organs and glands, where intracellular masses of blue cytoplasm studded with minute red granules represent schizonts which are known as Koch's blue bodies. These

break up into very numerous minute merozoites which re-enter other endothelial cells or parasitize the circulating blood as the before-mentioned gametocytes.

When infected blood is taken up by the vector tick, whether adult, nymph, or larva, development of the parasite takes place, with the production of sporozoites. Now, each of these stages of the tick is passed on a separate host, and the parasites taken up by one stage are transmitted to another host by the next succeeding stage of the tick. The adult tick, when infected, does not transmit the infection through eggs to the next generation of larvae, as happens in the case of various species of *Babesia* such as the one responsible for redwater fever.

This is the explanation of some of the anomalies found when the two infections are compared. Thus every egg laid by a redwater-infected female tick produces, when hatched out, a larva which can at once infect another animal. On the other hand, eggs deposited by ucks infected with East Coast fever produce uninfected larvae, which have to feed on an infected animal before they become infected. After their first feed they drop off the animal and attain the nymphal stage, in which they, in their turn, are infective. The adult female dies after laying eggs. In this way a very few cattle infected with East Coast fever can provide infection for many thousands of larval ticks, which in the nymphal stage are infective. The symptoms of East Coast fever begin 10–20 days after exposure to infection and start with fever. Lymphatic glands become enlarged and there is oedema of the subcutaneous tissues, intestinal mucosa, and serous membranes, with haemorrhagic patches. Calves are less susceptible than adults. The fever continues until death or recovery.

East Coast fever has existed, certainly for centuries, in East Africa, and was well known to the natives before it forced itself on our attention. It is most prevalent in well-grassed areas with high rainfall, and absent or sporadic in arid districts. It is widely distributed on the eastern side of Africa, but appears to be absent on the western side.

Mortality Due to East Coast Fever

It is difficult to get reliable figures on this point, and, in any case, it would seem probable that mortality varies in different districts and in different classes of cattle—European and indigenous. This will be evident from the opinions given by different observers: (1) East Coast fever is the cause of more deaths in cattle than all other causes put together; (2) the calf mortality from East Coast fever varies between 5 and 50%; (3) in South Africa the mortality from the natural disease is 95%; (4) deaths in bovines, the property of Europeans alone, in Kenya for 12 months ending July 31, 1928, were 16,728 out of 55,421 deaths from all other causes.

All this goes to show that, whatever the actual mortality rate, East Coast fever, where it exists, is far and away the greatest parasitic killing disease among bovines.

Another way to assess the relative importance of East Coast fever is supplied by the records of laboratory examinations for diagnosis of cattle diseases. I have examined the records available to me of laboratory examinations made in Kenya, over a period of 26 years, for the diagnosis of disease-producing organisms in bovines. These I have recorded in tabular form, but it will serve the present purpose if I indicate totals only. These are as follows:

Proportion of Diagnoses of East Coast Fever to all Other Diagnoses in Bovines

Year	Total Positive Bovine Specimens Due to Disease-producing Organisms Sent for Examination	No. Positive to East Coast Fever or Other Piroplasms, or Suspicious
1909–10 ..	413	344
1910–11 ..	939	847
1911–12 ..	900	699
1915–16 ..	1,101	505
1916–17 ..	813	395
1917–18 ..	550	357
1919–20 ..	829	671
1921	1,321	938
1922	1,340	475
1923	1,153	456
1924	1,407	769
1925	2,586	1,594
1926	4,808	1,969
1927	2,670	1,929
1928	2,072	1,640
1929	1,445	1,008
1933	1,581	865
1934	1,426	774
1935	3,037	434
1936	3,286	741
1937	1,564	696
1938	1,877	947
1948	557	284
	37,675	19,337

This assessment would give the astonishing result that diseases due to piroplasms, chiefly East Coast fever, are responsible for 51.3% of all diagnoses of disease-producing organisms. Now, even this gives no real idea of the economic importance of East Coast fever. The figures mentioned are merely those of specimens sent for examination. It takes no account of the probably immensely greater number of cattle affected but from which no specimens were sent, and it takes no account of the fact that these figures are for Kenya only. The figures have also to be considered in conjunction with the actual numbers of cattle in the area before an idea can be gained of the economic impact of this disease. I am indebted to Dr. E. Beaumont, Director, Veterinary Services in Kenya, for an estimate of the cattle population of Kenya, based partly on actual census-taking and partly on estimates. This indicates a cattle population of 5,773,554. To add to the blackness of the picture, mortality is not the only cause of economic loss, because infected animals which do not die are often badly out of condition and may remain stunted for a long time. This account might make you wonder how catttle exist at all on the east side of Africa. The fact is, of course, that areas of prevalence of East Coast fever are not continuous, nor are they all equally seriously affected.

Control

Methods of control do not strictly come within the scope of my address, but must be touched upon in general terms; and I do not propose to say anything at all about treatment of the disease.

Control measures are naturally directed against the ticks which are the vectors of the disease, and it is important to know that these measures can be very effective when properly applied and maintained. To put it in a few words, these methods are dipping, hand dressing, and fencing. These measures, carried out under strict supervision, are economically sound on valuable dairying land and can reduce the tick population over very large farms to proportions that are no longer a menace to the herds.

Conclusion

Man in his own estimation is the most important and culminating result of evolution in nature. How far this may be from the truth past events in the evolution of living things, which for a time dominated the earth and passed away, has shown, and as for the future, we cannot see it, so it behoves us to take a humble view, especially in the light of the most recent achievements of *Homo sapiens*. From our immediate point of view, however, we must consider man as just one unit in the vast complex of nature and realize that too great success may be disastrous. If he eliminates all the pests and diseases which afflict him he may multiply and eat up the earth and so destroy himself as the result of his success.

For these reasons, I would like to sound a note of warning against the danger that pests such as I have described should be treated, when preventive measures are considered, as if they produced their harmful results without reference to accompanying factors in their natural surroundings. Control measures must be devised in relation not only to destruction of a specific pest but in relation to all other circumstances which might follow the elimination of a single important unit in an ecological complex. Thus if the object in some areas is the complete removal of trypanosomiasis or piroplasmosis we must first consider whether this would not involve the production of a still more serious problem of soil erosion. Preventive or eradicative measures in such a case would have to be carefully planned in collaboration with experts on land usage *before* being put into operation, or we might find ourselves in the position of regretting the results of successful eradication when irreparable damage to the land had supervened.

In other words, an all-round approach must be made, and only after careful preparatory research should important measures of control be undertaken.

It must be remembered that in the case of bovines, which alone I have been able to consider, the chief evil at present is overstocking, and this is due to limitation of grazing lands by parasites of one sort or another. The scientific way of approaching the problem when eradication of pests is undertaken would be, in the first place, to use the method to reduce overstocking in areas already free of the particular pest or pests, and only then to try to increase the total head of cattle by extending still further the use of pasturage made safe by the eradication measures.

The theme, therefore, on which I would wish to close my address is a quotation from the report by H. E. Hornby on *Animal Trypanosomiasis in East Africa, 1949*, because I am sure that he would agree that his words have a wider significance than the context in which they were used. The quotation is: "The point to be driven home is that animal husbandry demands both pasture management and disease control. Improvement of pastures often encourages parasites by giving these protection from the sun, therefore it must be accompanied by disease control. This alone leads to overstocking, so it must be accompanied by pasture management to save the soil."

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RADIATION CANCER OF THE PHARYNX

BY

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There have been many examples of radiation cancer of the skin, but only a limited number of cases have so far been reported in which a tumour has developed in the deep tissues as a sequel to external therapeutic irradiation. The case histories of four patients who developed tumours in the laryngopharynx many years after irradiation of the neck may therefore be of some interest.

The majority of radiation tumours in the deep tissues have been sarcomata, which have arisen in bone. Hatcher (1945) reported three cases of sarcoma developing in irradiated bone, and reviewed the literature on the subject. He found a total of 24 cases. In 17 of these the condition primarily treated by irradiation was tuberculous arthritis, and the interval between irradiation and the appearance of a tumour varied between 3 and 12 years. There was a tendency for these tumours to develop when irradiation had been carried to excess in the form of repeated doses administered over a long period of time.

The only other cases in which cancer has been recorded as developing in the deep tissues after external therapeutic irradiation may conveniently be considered together, since their sites of origin lie within a somewhat limited anatomical field. Four of these tumours occurred in the laryngopharynx, one arose in the larynx, and one in the thyroid gland.

The first case was reported by Lossen (1936). A man was given *x*-ray treatment from 1919 to 1922 for tuberculous glands in the neck. In 1929 he developed a necrotic ulcer in the skin of the neck, and in 1935, 13 years after treatment, he developed a carcinoma of the larynx.

Kruchen (1937) reported the case of a woman aged 65 who developed a tumour on the posterior wall of the hypopharynx 20 years after irradiation of the neck for a goitre. The tumour had the structure of a carcinoma simplex. The skin on the anterior aspect of her neck was grossly telangiectatic.

Kindler (1944) reported two further cases. In the first of these a man of 54 developed an adenocarcinoma in the left lobe of the thyroid gland 24 years after *x*-ray treatment for tuberculous glands in the neck. He had marked post-radiation atrophy and scarring in the overlying skin and subcutaneous tissues. His second case was that of a woman of 59 who developed a tumour almost completely surrounding the pharynx at the post-cricoid level 20 years after irradiation of her neck for thyrotoxicosis.

Den Hoed (1946) reported two cases he had seen, both within the space of a year. The first was that of a man aged 43 who developed a carcinoma of the aryepiglottic fold 27 years after irradiation of his neck for tuberculous adenitis. He had a necrotic ulcer in the irradiated area of skin. The second case was that of a woman of 41 who, 28 years after irradiation for tuberculous glands in the neck, developed a squamous-celled carcinoma involving the aryepiglottic fold and arytenoid cartilage. Three years before this a basal-celled carcinoma behind the ear and within the irradiated area had been excised.

The following patients were all seen in this department within the space of eighteen months, and came from various parts of the area served by this Centre. They were given *x*-ray treatment many years ago, and it has not been possible to obtain details of dosage or other factors relevant to irradiation.

Case Reports

Case 1.—A woman aged 69 was seen in March, 1949, with the complaint that her voice had started to become hoarse three months before. The only other symptoms she had were related to radiation dermatitis of the neck, which had been giving her trouble on and off for at least 20 years. In 1923 she had been given a course of *x*-ray therapy for thyrotoxicosis. The skin overlying the thyroid cartilage was telangiectatic, and the subcutaneous tissues were fibrotic. On direct laryngoscopy she was found to have a friable tumour on the medial wall of the left pyriform fossa with extension on to the aryepiglottic fold on the same side. Section showed this to be a squamous-celled carcinoma. The interval between irradiation and the appearance of a tumour was 26 years.

Case 2.—A woman aged 50 was seen in February, 1950, with a history of dysphagia, which had been progressive for the past year. In 1920, when 21 years of age, she was given a course of *x*-ray treatment for thyrotoxicosis. She was uncertain how long the treatment had lasted, but it was probably between nine months and a year. In 1937 she developed what she described as a growth in the skin immediately over the thyroid gland. This was excised and repaired with a skin graft. In the absence of a histological report it is not known whether the condition was malignant or necrotic. After this she remained well until the onset of her present symptoms. On examination she had a grafted area about 10 cm. square over the thyroid gland. Direct laryngoscopy revealed a tumour in the post-cricoid region, extending down to involve the first 2.5 cm. of the oesophagus and superiorly involving the pharyngeal surface of the aryepiglottic fold. Section showed the structure of the tumour to be predominantly that of a fibrosarcoma. The interval between irradiation and the appearance of a tumour was 29 years.

Case 3.—A woman aged 76 was seen in April, 1950, the presenting symptom being dysphagia. This had first been noticed about 18 months before, and had become progressively worse since its onset. More recently she had developed referred pain in the right ear. She had received *x*-ray treatment for thyrotoxicosis in 1922. She stated that treatment had been given once a week for a period of six months. In 1936 she had had a skin graft performed on the anterior part of her neck. On examination she had a hard tender gland in the upper part of the deep cervical chain on the left side, and there was a grafted area of skin over the thyroid cartilage. Oesophagoscopy revealed a granular bleeding stricture in the immediate post-cricoid region. Section showed the tumour to be a squamous-celled carcinoma. The interval between irradiation and the appearance of a tumour was 27 years.

Case 4.—A woman aged 49 was seen in August, 1950, and gave a history of having developed a sore throat about seven months before. This was associated with the sensation of having a lump in her throat, which she could not swallow. More recently she had developed referred pain in the right ear. In 1919, at the age of 20, some tuberculous glands had been removed from the right side of her neck. After this she was given prolonged *x*-ray treatment to the neck. The total dose is not known, but treatment extended over a period of a year. On examination there were no obvious radiation changes in the skin of her neck. On direct laryngoscopy she was found to have a tumour arising from the posterior wall of the hypopharynx, just above and apparently involving the cricopharyngeal sphincter. Section of material removed at biopsy showed a highly infiltrative but moderately well-differentiated squamous-celled carcinoma. The interval between irradiation and the appearance of a tumour was 30 years.

Discussion

It may sometimes be difficult to decide whether a tumour in the deep tissues can be attributed to previous irradiation. Smith and Bowden (1948), in an attempt to determine whether uterine cancer followed the use of radium therapy for benign uterine conditions, investigated 805 proved cases of carcinoma of the body of the uterus. The criteria they adopted for the diagnosis of radiation cancer were (*a*) a ten-year interval between irradiation and the development of a uterine tumour; and (*b*) an originally confirmed negative endometrial biopsy. In only eight cases were these conditions satisfied. In the absence of any reliable figures for the natural incidence of uterine cancer they found it impossible to decide whether the irradiation of benign uterine conditions played any part in inducing cancer at a later date. It is suggested that the diagnosis of radiation cancer should be based on the following considerations.

1. It cannot be assumed that cancer has developed in response to previous irradiation unless this was used primarily in the treatment of a benign condition.

2. There must be a latent interval between irradiation and the development of a tumour. This has been a characteristic feature of all radiation tumours, and must therefore be regarded as an essential factor in the pathogenesis of radiation cancer. In a series of patients with skin cancer following therapeutic irradiation who were seen at the Radium Institute in Paris, the latent interval varied between 4 and 21 years (Lacassagne, 1945). The two cases reported by Walter (1950), in which epitheliomata developed in the skin of the neck within three months of irradiation, are without precedent and hard to explain.

3. It seems probable that neoplastic change occurs only in tissues which have suffered appreciable radiation damage. Skin cancer supervenes typically in an area of skin showing obvious radiation changes, and radiation tumours which have developed in the deep tissues have been accompanied by gross changes in the overlying skin and subcutaneous tissues.

4. There should be histological proof of any tumour which is suspected of arising as a result of irradiation. A late necrosis might simulate malignant disease.

5. Radiation tumours often display some abnormality in respect of sex and age incidence, anatomical site, or microscopical appearance when these are considered either separately or in relation to one another. It is not uncommon to find atypical forms of squamous carcinoma in radiation cancer of the skin, and tumours in this situation may be fibrosarcomatous or have a pseudosarcomatous appearance (Lacassagne, 1945). The incidence of chondrosarcomata is high in tumours which have developed in irradiated bone (Hatcher, 1945). The way in which age, sex, and site may indicate the nature of a tumour is well illustrated in one of den Hoed's cases where a carcinoma of the aryepiglottic fold occurred in a woman in the fourth decade of life. As he points out, this is exceptional.

The four patients whose case histories are presented above have certain features in common, apart from their site of origin. In all of them neoplastic change occurred after a long latent interval, which varied only between 26 and 30 years. They were all treated originally for benign conditions, and in three of them it is known that frequently repeated doses of *x* rays were given over a period of six months or more. In the three patients treated for thyrotoxicosis the tumours had arisen approximately 4 cm. deep to the centre of the irradiated area of skin, and these three patients had all suffered severe radiation damage to the skin, two of them requiring excision and plastic repair for either epitheliomatous change or necrosis. Case 4 alone failed to show any convincing evidence of skin damage, and for this reason it is doubtful whether the tumour can be attributed to irradiation. However, this is a comparatively uncommon site for cancer, and it is difficult to be persuaded that its appearance was purely coincidental. Radiation is only one of many known carcinogenic agents, and, although it cannot be held solely responsible, it may well have been a contributory factor in the genesis of this tumour.

The remaining three patients all seem to have developed tumours as a direct result of irradiation, and in two of them there are features which further support this aetiology. In Case 2 the tumour had the structure of a fibrosarcoma, and, although this tumour may rarely occur in the upper part of the oesophagus, it must be very rare in post-cricoid tumours. As Cade (1951) has pointed out, fibrosarcoma may develop on a basis of post-radiation scarring. In Case 1 the tumour arose in the pyriform fossa, which may be significant, as carcinoma in this site is predominantly a disease of men.

In conclusion it seems reasonable to speculate that, if the treatment of thyrotoxicosis by intense or prolonged irradiation was a common practice 25 to 30 years ago, further cases of radiation cancer in the laryngopharynx may occur, or have perhaps already occurred.

Summary

It is rare to find a tumour in any of the deep tissues following external irradiation, and the literature on this subject is briefly reviewed.

The case histories of four patients who developed tumours in the laryngopharynx after irradiation of the neck are presented. The criteria considered to be important in the diagnosis of radiation cancer are enumerated, and the reasons for regarding three of these tumours as examples of post-radiation cancer are given. In the fourth patient the aetiology of the tumour is open to doubt.

I wish to thank Dr. A. G. C. Taylor for permission to publish these cases and for the encouragement, advice, and criticism he has given in the preparation of this paper. I am grateful to Dr. H. H. Gleave for histological details and also to Dr. T. V. Cooper, of Dorchester, and Dr. R. D. Clay, of Portsmouth, for histological details in two of these cases.

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FERROUS SULPHATE POISONING IN CHILDREN

BY

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Iron poisoning in children occurs through the temptation of attractive sugar-coated pills containing ferrous sulphate which, with good reason, are freely supplied to their mothers. The first comprehensive reports of this disaster in children were published in 1947, and, though it is difficult to believe that cases did not occur before, the first recorded case was that of a child of 16 months who died, in May, 1944, after swallowing 39 ferrous sulphate tablets (Thomson, 1947). Since this time there have been further case reports, and the clinical picture of the poisoning is becoming clear.

The daily press has recently given prominence to inquests on children who have died after swallowing ferrous sulphate tablets, and I have used this source of information to collect some details of four cases to add to the reports of four which I have seen myself. Brief reference has already been made to two of these coroners' reports (Cases 6 and 8) in a leading article in the *Lancet* (1949). The daily press has also made reference to the ignorance of parents and doctors alike concerning the effects and dangers of these tablets to small children.

It is the purpose of this paper to describe the clinical picture of iron poisoning in detail, to discuss the effects of large doses of iron, to propose a more positive therapeutic approach than has been given in previous medical articles, and to suggest a method of safeguarding children from these tablets.

The Tablets.—The most readily available tablets are green sugar-coated pills, and each of these contains ferrous sulphate, 3 gr. (0.2 g.), copper sulphate, 1/25 gr. (2.6 mg.), and manganese sulphate, 1/25 gr. (2.6 mg.). Forbes (1947) has shown by animal experiments that the effect of the copper and the manganese can be discounted, and that it is the ferrous sulphate which has the irritating and lethal effect.

The cases recorded below have, with the exception of the coroners' cases, all been treated is this department within the space of a year. In this period three other children were admitted with a diagnosis of ferrous sulphate poisoning, but they have been excluded because of lack of detail regarding what they had swallowed. One had the symptoms of poisoning, but the other two were not upset. The mother of one of the latter, aged 18 months, was quite sure he had swallowed 24 tablets, but only minute quantities of iron were recovered from the stools and stomach washings, and it was thought more probable that only a few pills had been taken than that 24 tablets had not made him ill.

In the seven preceding years during which this department has been in existence only one other such case has been admitted. This was a girl aged 2½ years who swallowed 60 tablets. Her mother thought she vomited back "nearly all the tablets," and, though she was pale and drowsy and had marked diarrhoea and vomiting, her recovery during the subsequent three days seems to

have aroused no special interest at the time. Her case has been excluded owing to the doubt about the number of tablets she retained.

Case 1

Between 5.30 and 6.10 p.m. on June 26, 1950, a girl aged 21 months swallowed 75 ferrous sulphate tablets. These made her sick, and her father, realizing what had happened, held her upside down over the kitchen sink, whereupon she vomited 21 tablets. After this a nurse gave her some salt and water to drink, and she was sick again. Later the family doctor washed her stomach out with normal saline, but on neither occasion were further tablets returned. From 6.15 p.m. the child was said to have been cold, pale, and semi-conscious.

At 8.35 p.m. she arrived in hospital, and her condition was that of a well-nourished but shocked child. She was cold and pale, with slightly cyanosed lips, and her pulse was rapid (150 a minute) and difficult to feel. She was semi-comatose in that she showed little resentment to painful stimuli, but she did open her eyes momentarily when roused. Further examination revealed no abnormality. Gastric lavage was carried out with normal saline, and 8 oz. (230 ml.) of sodium bicarbonate solution was left in the stomach. During this manipulation the returning fluid contained shreds of tissue and streaks of blood but no further tablets.

For the next 10 hours the child was unaware of her surroundings. She was very restless and frequently retched and vomited small quantities of dark red blood. Her respirations were irregular, shallow, and rapid (60–70 a minute) and her temperature rose to 102.8° F. (39.3° C.).

By 6.30 a.m. on June 27 she seemed a little better and was warm, though still pale and very restless. She was retching frequently but not vomiting quite so often, and for the remainder of the day her condition remained much the same. She occasionally showed a little interest in her surroundings and appeared to recognize her father. By evening she was drowsy and slept for short spells, but was very restless in the intervals. She was vomiting "coffee-ground" material but also taking drinks of water. Her bowels were opened three times, the stools being small, dark, and very offensive.

On June 28 she was taking a little more interest in her surroundings and tried to sit and stand up occasionally, but she was very "floppy" and seemed too weak to do so. She vomited "coffee-ground" material on two occasions, but was very drowsy and slept most of the time. Next day the improvement was maintained. She slept well through the night and was still drowsy during the day. No further vomiting occurred. On the 30th she was much more lively but still rather "floppy," and could not sit up on her own yet.

On July 3 her behaviour was normal, and she was discharged from hospital on July 7. A fortnight later her appetite was poor, and she had vomited at least once a day at home. Thereafter vomiting ceased, and on August 31 the mother said that her child "has really been back to her normal self for the past week."

Investigations.—June 26: 10.30 p.m., serum iron, 3.3 mg. per 100 ml. (normal, 0.035–0.22 mg. per 100 ml.). *June 27:* 1.30 a.m., serum iron, 2.25 mg. per 100 ml.; 9.30 a.m., serum iron, 1.12 mg. per 100 ml.; 10.15 a.m., B.P., 120/70; haemoglobin, 68%; 10.30 a.m., E.C.G., normal; 5 p.m., E.C.G., normal; 10.30 p.m., B.P., 105/60. *June 28:* 10 a.m., B.P., 135/70; haemoglobin, 60%; urinary iron content, *nil*; 3 p.m., urinary iron content, *nil*. *June 29:* 10 a.m., B.P., 120/65; haemoglobin, 62%; E.C.G. normal; 12.30 p.m., serum iron, 0.26 mg. per 100 ml.; urinary iron, 0.45 mg. per 24 hours. The urine contained a trace of albumin; microscopy showed nothing abnormal. *August 31:*—Liver-function tests: zinc sulphate, 9 units; thymol turbidity, 12 units; thymol flocculation, +++; cephalin-cholesterol, +++; alkaline phosphatase, 23.8 units.

Case 2

At 11 a.m. on February 2, 1951, a boy aged 23 months was found to have eaten 16 ferrous sulphate tablets and 10 iron plastules (the total iron content of tablets and plastules was 98 gr. (6.5 g.) of ferrous sulphate, or the equivalent of 33 ferrous sulphate tablets). He was immediately given salt and water to drink, and he vomited some "black liquid slime" containing some partly dissolved plastules and four tablets. In the next hour he vomited several times and then went to sleep. When he awoke at 1.45 p.m. he vomited about a tablespoonful of bright red blood, and at 2.15 p.m. he was admitted to hospital.

Examination showed a boy of average physique; he was pale and had cold extremities, and was retching frequently. His pulse rate was 140, his blood pressure 90/60, and his respiration rate 35. For the next 10 hours retching and vomiting continued, and about every half-hour he brought up a little blood and sometimes shreds of mucosa. He was restless and quiet in turns, but was co-operative and quite aware of his surroundings. At 11 p.m. both plantar responses were extensor.

For the next 24 hours he remained drowsy and slept fitfully, but when awake was still very restless. Vomiting was not so frequent and no longer contained blood, and after midday it ceased altogether. His general condition improved and his extremities were warm, but both plantar responses remained extensor.

On February 4 alternating drowsiness and restlessness continued, but he appeared brighter in himself and took more interest in his surroundings. His temperature had gradually risen to 101° F. (38.3° C.), but thereafter was normal. Next day his behaviour was normal, plantar responses were flexor, and his bowels were opened for the first time. He was discharged home on February 8, and two weeks later was "very well."

Investigations.—February 2: 3.30 p.m., serum iron, 3.42 mg. per 100 ml. *February 8:* serum iron, 0.33 mg. Liver-function tests: thymol turbidity, 3 units; thymol flocculation, *nil*; cephalin-cholesterol, +; zinc sulphate, 3 units; alkaline phosphatase, 5.8 units; serum bilirubin, absent.

Case 3

At 5.30 p.m. on November 25, 1950, a boy aged 11 months was found vomiting on the floor and beside him was a packet of ferrous sulphate tablets: 13 tablets were missing. He was put to bed but continued to vomit, and the parents estimated that he had returned bits of tablets amounting to four to six whole tablets. He thus retained seven to nine tablets. By 6 p.m. he had turned very pale and lay very still, crying only when he wanted to vomit. The vomit was a clear brown fluid and did not contain blood. On admission to hospital at 6.40 p.m. he appeared pale, quiet, and rather drowsy, and there was slight cyanosis of the lips. At 7 p.m. the stomach was washed out; at first he cried and struggled, but towards the end of the procedure he lay very still. Physical examination at this stage showed a well-nourished infant with no abnormal signs. The pulse rate was 150, the respiration rate 35, and all deep reflexes were present and brisk. For the next three hours he remained very quiet and pale, and he vomited once. Thereafter his colour improved and he slept through the night. The next day he appeared lively and quite normal. He passed two loose black motions; the pulse rate dropped to 100 and the respiration rate to 24. On November 27 he remained well, and next day was discharged home.

Case 4

At 3.30 p.m. on February 6, 1951, a boy aged 20 months was noticed by his mother to be rather quiet; she thought he looked drowsy and ill, and she became alarmed when she could not rouse him properly. His sister said he had been eating some tablets, and investigation showed that a maximum of five ferrous sulphate tablets were missing. At 4.30 p.m. his mother gave him some salt and water to drink,

and be vomited several times, two tablets being returned. At 6.30 p.m. he was admitted to hospital and kept under observation for 24 hours, but he was cheerful and lively on arrival, and at no time showed any ill effects from the three tablets which he had retained. Treatment was considered unnecessary.

"Coroners' Cases"

Case 5

At 12.30 p.m. on August 20, 1950, a boy aged 12 months, who had always been healthy, was put to sleep in his parents' bed. At 1.15 p.m. he was found playing with an unknown quantity of ferrous sulphate tablets, and four or five were removed from his mouth. He was made to drink salt and water and he vomited, but no tablets were returned. He was given some castor oil and then ate his dinner. He did not appear to be upset. At about 2.45 p.m. he vomited a brown-coloured liquid containing six tablets. The family doctor prescribed a further dose of castor oil, and said that if the child became worse he should be sent to hospital. The child went to sleep but woke up again and vomited. He was cold and clammy. At 5 p.m. he was found to be dead.

Necropsy Report.—The body was that of a well-developed male infant. The nostrils, lips, and mouth were stained with a black liquid. The nasopharynx, trachea, and main bronchi contained much brownish-black semi-solid material, which was not frothy, and the lungs showed some mottling on their surfaces due to scattered areas of collapse. The stomach contained about 3 oz. (85 ml.) of thick black fluid and the mucous membrane was red and necrotic. A few scattered areas of congestion were present in the upper jejunum, and Peyer's patches of the entire small intestine were prominent. The large intestine contained black fluid but was otherwise normal. The liver was normal in size but rather pale. The heart, spleen, pancreas, kidneys, skull, and brain were normal macroscopically. The stomach contents gave a strongly positive dipyridyl reaction for iron.

Histology.—The stomach showed necrosis of the superficial layers of the mucosa. Prussian-blue staining revealed a mass of iron in the necrotic portions of the mucous membrane and smaller amounts being absorbed into the blood stream. The liver showed cloudy swelling of the cells of the centrilobular zone and a slight increase of polymorphonuclear leucocytes in the liver sinusoids.

Case 6

At 8.15 a.m. on September 10, 1947, a girl aged 19 months was found vomiting after taking 15 or 16 ferrous sulphate tablets. She was taken to hospital and there given salt and water, and she vomited again. The mother was told that there were no beds available, and she was sent to another hospital. Here she was told that the tablets were not poisonous and would do the child no harm. The child was retching now but not vomiting, and the mother was told to take her home and give her plenty of milk to drink. The mother was not satisfied and took the child to another doctor on the way home. He told her to give the child orange juice to drink and she would be all right. The mother then took the child home, put her in her cot, and went to make some orange juice. When she returned the child was dead. The time was 1 p.m., about four hours after the child had taken the tablets.

The necropsy showed intense congestion of the stomach, which contained blood and mucus, and the intestines were also congested. There was no vomit in the respiratory passages.

Case 7

At 9 a.m. on January 4, 1951, a boy aged 18 months started to cry and was given a cup of tea, whereupon he was violently sick and had diarrhoea. His mother found her box of ferrous sulphate tablets empty, and 44 tablets were missing. He was taken to hospital, where his stomach was washed out with 5 pints (2.8 litres) of fluid, flecks of blood and mucus being returned. Restoratives were administered, but he died. He was said to have been in a state of coma from 11 a.m., and he died at 2.30 p.m., five and a half hours after taking the tablets.

Necropsy showed acute dilatation of the right ventricle and congestion of the stomach and intestines, with, on analysis, substantial quantities of iron in the stomach contents. There was no vomit in the respiratory passages.

Case 8

One morning in September, 1949, a girl aged 14 months was found to have swallowed 44 ferrous sulphate tablets. The doctor was telephoned, but he said there was no danger. Later the child showed signs of distress and started to vomit, bringing up four tablets. The doctor was sent for and found a rapid pulse. He again said that there was no danger, but he called back later and prescribed castor-oil and kaolin. The child vomited and retched throughout the day, and in the evening was said to be semi-conscious and was put to sleep in her parents' room. During the early hours of the following morning the mother found her child to be dead, some 20 to 24 hours after taking the tablets.

At necropsy the stomach contained a quantity of black iron sulphide and showed intense congestion and oedema of its wall, with, in places, corrosion of the mucosa. There were "profound toxic changes in the liver."

The Clinical Picture

Excessive amounts of ferrous sulphate have a fairly constant effect on small children. Within the first hour even the mildest cases make their mothers apprehensive, for they look pale and ill and they generally vomit. At first the vomitus may contain unaltered tablets, and in the more severe cases it often contains small amounts of bright red blood by the third hour, and by this time the child presents the fully developed and characteristic

Summary of Case Records

Case No.	Age (Mths)	Sex	No. of Tablets Taken	No. of Tablets Returned and After How Long	No. of Tablets Retained	Treatment Before Admission	Time Before Admission	Treatment After Admission	Tachycardia	Early Pallor	Drowsiness	Vomiting	Haematemesis	Result	Time Before Death
1	21	F	75	21. 30 mins.	54	Salt water; gastric lavage	3 hours	Gastric lavage; bismuth carb.; vitamin mixture	+	+	+	+	+	Recovered	—
2	23	M	33	4+. Few mins.	Less than 29	Salt water	3½ ,,	Bismuth carb.; I.V. glucose-saline; vitamin mixture	+	+	+	+	+	,,	—
3	11	M	13	4–6. Few mins.	7–9	Nil	70 mins.	Gastric lavage; bismuth carb.; vitamin mixture	+	+	+	+	—	,,	—
4	20	M	5	2. 1 hour	3	Salt water	3 hours	Nil	—	—	+	—	—	,,	—
5	12	M	?	6. 1½ hours	?	Salt water; castor oil	—	,,	?	?	+	+	—	Died	4 hours
6	19	F	15–16	None	15–16	Salt water	—	,,	?	?	?	+	—	,,	4 ,,
7	18	M	44	,,	44	Nil	?	Gastric lavage	?	?	+	+	—	,,	5½ ,,
8	14	F	44	4. ?	40	Castor oil; kaolin	—	Nil	+	?	?	+	—	,,	20–24 hours

This table is similar to that used by Thomson, but has been slightly amended.

picture. Pallor, coldness, tachycardia, retching, vomiting, and drowsiness, together with restlessness, are almost constant. Of these vomiting and drowsy restlessness are the predominant features of the illness, and the length of time they continue depends on the number of tablets taken. Thus Case 4 was drowsy and looked ill for an hour or two after only three tablets, whereas Case 1, who retained 54 tablets, was semi-comatose for 24 hours and drowsy for four days, and it was eight days before she behaved normally.

Haematemesis in the first 12–24 hours is frequent, and, though it is an alarming symptom, it does not usually lead to an excessive loss of blood. Diarrhoea is uncommon, which is surprising when one considers the irritating effect of the tablets on the stomach. However, the small bowel escapes gross damage, and this may be due to the reaction of the alkaline intestinal juices converting the ferrous sulphate into insoluble iron compounds.

Increase in the respiratory rate was noticed in the first three cases, and in Case 1 the alteration was pronounced, the excursions were very shallow, and the rhythm was also irregular. These features have not been reported in other cases, though Somers (1947) noticed shallow breathing and an increased rate in experiments on rabbits. Two other cases have been reported with abnormal physical signs in the chest, but, in these, aspiration pneumonia was the probable cause.

Detailed physical examination adds little to what has been noted already. Abdominal distension is not present and tenderness is only infrequently found, and then is of slight degree. The central nervous system, apart from the altered mental state, has shown abnormal signs only in Case 2, in which bilateral extensor plantar responses were noticed on the first evening, and these reverted to normal on the fourth day as the child recovered.

A further feature that must be mentioned is a misleading period of clinical improvement preceding collapse which has been observed in some fatal cases during the second and third 12-hour periods. Thus Thomson's (1950) fourth case (reported fully by Prain, 1949) appeared quite well on the second day, but collapsed quite suddenly and died after 39 hours, whilst the other death in Thomson's series and Forbes's first case also followed this pattern.

Study of the case records of the eight deaths so far reported (including those in this series) reveal two critical periods in the illness. The first is after four to six hours, when three of the children died; the second is from 20–53 hours, and it includes the remaining five deaths. The significance of these periods is discussed later.

Mode of Action

It is clear from the necropsy findings in all cases that the stomach bears the brunt of the initial damage. It is reported as being oedematous and congested, with haemorrhagic and necrotic areas of variable extent which mainly involve the crests of the rugae. The small intestine is generally affected only in its proximal part, and then to a less degree than the stomach, and it shows congestion and perhaps oedema of the mucous membrane. The liver also shows gross changes, which vary from cloudy swelling to areas of necrosis, but the only other abnormalities that have been detected are cloudy swelling of the kidneys, heart, and pancreas, congestion of the spleen with necrosis of the Malpighian corpuscles, and congestion and patchy collapse of the lungs with aspiration pneumonia.

Prain suggested that liver failure is the cause of death in iron poisoning, but he admits that "the extent and degree of change in the liver seem scarcely sufficient to explain the fatal issue." It seems clear, too, that if liver failure is to be blamed for death then the histological changes are hardly comparable to those found in, for example, acute yellow atrophy, and in iron poisoning death occurs much quicker than is usual in that disease. For these reasons I would suggest that one must look elsewhere for the cause of death.

Serum iron estimations in Cases 1 and 2, between four and five hours after the tablets had been swallowed, showed levels of 3.3 and 3.4 mg. of iron per 100 ml., compared with the normal of 0.035–0.22 mg. per 100 ml. (Sven Dahl, 1948). These figures approximate almost exactly to the level, quoted by Slack and Wilkinson (1949), of 3.6 mg. per 100 ml. which is attained after an intravenous injection in adults of 200–300 mg. of an iron sucrose preparation. The symptoms reported to follow intravenous injections of iron include pallor, headache, vomiting, weakness, and collapse (Ramsey, 1950), and encephalopathy (Birch and Till, 1951), while Hurst (1931) and Napier (1936) recorded cases of presumed iron encephalopathy in adults after iron medication by mouth. There would seem to be a similarity here between the effects of iron given intravenously in adults and poisoning by ferrous sulphate in children, and I feel that iron poisoning results in a widespread interference with cell function, and that probably the most important organ involved is the brain. Histological proof of this is unfortunately lacking, for the available necropsy reports do not mention detailed examination of the brain.

I would suggest that the genesis of the illness is as follows. The initial vomiting, haematemesis, tachycardia, and collapse are due to the direct corrosive effect of the iron on the stomach. This causes considerable shock (as in other corrosive poisons), and it is this, together with the rising serum iron, which is the main cause of death in the first period (four to six hours). This stage of shock lasts for some 12–24 hours and then passes off, and it is its passing which causes the misleading improvement seen in some fatal cases during the second and third 12-hour periods. Concomitant with this process, and starting very soon after swallowing the tablets, iron is absorbed into the general circulation, and the serum iron may reach 15–100 times the normal level. The necropsy findings, though incomplete, make it reasonable to suppose that this amount of iron can cause profound cell dysfunction and that its effect will be general, though some tissues will be damaged more than others. The occasional untoward sequelae of intravenous injections of iron in adults and the not dissimilar type of illness observed in severe iron poisoning in children suggest that the central nervous system is deeply involved, and I feel that iron probably exerts its most important effect on the cells of this system and through this action causes death.

Treatment

Hitherto all reported cases have been treated "on general principles." That is to say, the stomach has been emptied either by an emetic, such as salt and water, or by lavage, and bicarbonate of soda or bismuth carbonate, or both, have been utilized to convert the soluble ferrous sulphate into insoluble iron compounds. These measures are both rational and simple, and were carried out on the cases in this series. In Case 5 six undissolved

tablets were vomited one and a half hours after the child had taken them, so, clearly, every effort should be made to empty the stomach in the first few hours; and it should be remembered that whole tablets will not pass through a stomach tube.

Dimercaprol ("B.A.L.") has been used on two occasions (Thomson, 1950; Roxburgh, 1949), but no claim of its efficacy has been made, and Edge and Somers (1948) have shown in experimental work on mice that when used either orally or intravenously it increases, rather than decreases, the toxic effects of ferrous sulphate. It was not used in this series.

Shortly after the admission of Case 1 to hospital Dr. A. L. Latner, of the Department of Biochemistry of this hospital, was called in consultation, and he made the following suggestions: (1) As iron might act as a heavy metal and combine with SH groupings and thereby interfere with oxidation, tocopherol, which acts as an antoxidant and appears to cut down cell oxidative requirements, should be prescribed. (2) Methionine should be given (*a*) as a source of SH groupings and (*b*) in an attempt to prevent the development of the fatty changes in the liver seen in some previous cases. (3) That the drowsiness and sudden death of previous cases might be due to some interference with the oxidative enzymes and with the utilization of the vitamin B complex. Also, as deficiency of certain members of this group is associated with fatty change in the liver, then members of the vitamin B complex should be given as well.

The outcome of these suggestions, later known as the "vitamin mixture," was prescribed, and the ultimate recovery of Case 1, who had taken a larger dose of iron than any other case previously described, encouraged us to use a similar regime in Cases 2 and 3, with equally satisfactory results. In Case 1 this treatment was started 22 hours after the tablets had been swallowed, and in Cases 2 and 3 it was started within a few hours of the child's arrival in hospital.

When Case 2 arrived in hospital, also having taken a very large dose of ferrous sulphate—98 gr. (6.5 g.)—though in this instance not all in tablet form, it was argued that if the 20–53-hour period was a critical one then it was important to see that the child entered it in as good general condition as possible. This child had haematemesis and was vomiting and thirsty, but he was not clinically dehydrated; however, he was given a slow intravenous infusion of 5% glucose-saline in order to maintain rather than correct the fluid balance. After this was carried out his condition improved, he had no further vomiting, and he made a rapid recovery.

The cases in this series and those described by Thomson (1947, 1950), Forbes (1947), and Roxburgh (1949), with a total mortality of 47% (17 cases, 8 deaths), give a more depressing view of ferrous sulphate poisoning than is justified, for there must be many mild cases which require little or no attention and, because they arouse no particular interest, are not reported. However, while these tablets remain in such common use it seems only too certain that there will be further cases, and for these I would suggest the following treatment:

1. Efforts to make the child vomit should be carried out immediately in the home, either by giving him salt and water to drink or by placing the fingers in his throat.

2. Gastric lavage with sodium bicarbonate solution as soon as possible: 10 oz. (285 ml.) of the solution should be left in the stomach.

3. Bismuth carbonate, 3 gr. (0.2 g.) should be given four-hourly.

4. Precautions should be taken against the inhalation of vomit.

5. Intravenous infusion of fluids should be considered. If shock is severe plasma should probably be used initially, otherwise 5% glucose-saline should be given to maintain fluid balance in severe cases.

6. The following vitamin mixture should be given:

Aneurin hydrochloride	10 mg.
Nicotinamide	30 „
Riboflavin	10 „
Tocopherol	15 „
Methionine	500 „

These amounts should be multiplied by the child's age in years and then divided into three daily doses.

With the exception of methionine all these substances can be given intramuscularly until vomiting stops; thereafter they can be given by mouth and often in tablet form, for these children have already shown an aptitude for swallowing tablets.

Prevention

Following, perhaps, on the suggestions of others who have written on this subject, the leading manufacturers of ferrous sulphate tablets have now printed in large letters on their packets that these tablets are dangerous to young children. This is the first step in prevention, but so far it is the only one that has been taken. Now, it seems irrational that some things which children are expected to take, such as cod-liver oil, remain unpalatable, whereas ferrous sulphate tablets, which are intended mainly for adult use, are made both attractive and sweet. Possibly adults would not take them if they were otherwise, and no suggestion would be acceptable which had this result; for these tablets play a vital part in maintaining the health of large numbers of the population, including the pregnant woman. For this reason I feel that the tablets should remain in their present form, but that some obstacle should be placed between the tablet and the child, and I would like to suggest that this could be done as follows:

The daily dose of three tablets should be wrapped separately and securely in small packets in the same way that sugar is sometimes supplied in hotels in packets containing two lumps. In the German Army Medical Service nearly all tablets were supplied in packets of five, and 10 of these units were fixed together, making bundles of 50. This was done presumably for ease in handling, but if it could be carried out as a general measure it should also be practicable in this instance. A glance at the case histories shows that almost all the children poisoned are under 2 years of age, and I feel that if members of this age group accidentally obtained tablets wrapped in the manner suggested they might play with them for a long time without opening the packet, and, even if they did free some of the pills, the speed at which they could swallow them in quantity would be very effectively reduced.

Summary

The clinical picture of iron poisoning in children is described in detail.

The effects of large doses of iron on children, and the genesis of the resulting illness, are discussed.

A new method of treatment is proposed.

A suggestion is made whereby further cases might be prevented.

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CHILDHOOD FACTORS IN DUODENAL ULCER*

BY

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At the present time it is a commonplace to regard duodenal ulcer as a "psychosomatic" illness. By this most authors imply that it is a disease which is caused, in part at least, by psychological stresses to which, according to many, a number of people are predisposed by a specific type of personality.

While there is a certain amount of agreement on the nature of this personality among the various authors who believe in a psychosomatic aetiology of the disease, very few attempts have been made to demonstrate that patients with duodenal ulcer differ significantly from the general population. The difficulties of making such a comparison are great, as "personality" is an intangible quality and not easily measured. Moreover, the work of those who have endeavoured to overcome these difficulties—for example, Davies and Wilson (1937) and Brown *et al.* (1950)—is open to the objection that any difference shown between a group of ulcer patients and a group of controls may be due to the effects of the disease rather than be an indication of its cause : it is conceivable that chronic pain in itself may affect the personality, or at any rate the performance of psychological tests.

It is therefore surprising that the childhood of patients with duodenal ulcer has not been more extensively studied, as any difference between the childhood of these people and others could not be the result of the disease. All psychologists are emphatic on the importance of the childhood in the genesis of the adult personality. Moreover, there are a number of clear-cut factors in childhood of importance in the development of personality which can be assessed accurately and used for a numerical comparison between groups.

A few authors have investigated the problem of duodenal ulcers from this aspect. Draper and Touraine (1932) found more males than females among the siblings of duodenal ulcer patients in the proportion of 138 males to 100 females, in contrast with a group of cases of gall-bladder disease, in which the female siblings outnumbered the males in the proportion of 130 to 100. This was based on an investigation of 32 cases of peptic ulcer and 32 of gall-bladder disease. They use this observation to support their theory of a masculine–feminine conflict in ulcer patients.

Mittelmann and Wolff (1942), in a review of 30 cases of duodenal ulcer, conclude: "The failure of the home in every instance to lend a stable background resulted from a variety of causes—unhappy married life of parents, separation or loss of father during the child's development, early remarriage of mother after separation from father."

Kapp, Rosenbaum, and Romano (1947) also state : "In each patient the origin of such strong dependent wishes was traced either to rejection in childhood or to spoiling during the same period."

Moses (1946) also considered the childhood important. He writes : "This marked basic insecurity was the most prominent feature common to the entire group. It was related to the concrete frustrating economic and emotional environment in which they were reared. The majority of these individuals came from broken homes and from a marginal economic level with its limited social and educational opportunities."

Ruesch (1948), comparing duodenal ulcers in 42 naval personnel and 20 civilians, makes the following statements : "Ulcer bearers were generally the youngest or the younger-born children in the family. In terms of years they were separated from their older and younger siblings. . . . Our patients were on the average between 3 and 6 years old at the birth of a subsequent child. . . . The naval patients were characterized by a preponderance of brothers and the civilian patients by a greater frequency of sisters."

Thus it will be seen that there is little agreement among those who have investigated this problem, and most of them have considered small series of cases. Therefore it seemed worthwhile to compare a fairly large group of patients suffering from duodenal ulcer with a similar group suffering from other diseases. Gastric ulcers were excluded in order to reduce the number of variable factors, and because most authors in the past have considered psychological factors to be of more importance in duodenal ulcer than in gastric ulcer.

Present Investigation

The points chosen for investigation were those factors in childhood which might be of importance in affecting the personality and which could be ascertained with accuracy—for example, size and composition of family, age at death of parents, illness in childhood, etc.

Factors believed to affect the incidence of duodenal ulcer are sex, age, occupation, geographical situation, and time. To eliminate errors due to these factors the investigation was limited to males, and the patients in the control group were chosen to match the ulcer group in five-year age groups.

Occupational differences can only have been slight, as all were hospital patients in a regional hospital in North-west London, and, as is shown later, there was no difference between the two groups in social class. Temporal differences can be ignored, as both the ulcer patients and the control group were interviewed in 1950.

*This paper is based on a thesis for the degree of M.D.Cantab.

Interviews were had with 250 patients with duodenal ulcer and the same number of patients suffering from various other diseases, who acted as controls. The last 164 in each group were interviewed more fully in order to extend the scope of the investigation, but there was no particular selection of these, as all the ulcer patients were consecutive, except that there was an interval after the first hundred. Only one ulcer patient was not included—a hemiplegic following a birth injury who was mentally defective and incapable of answering questions. The age distribution of the ulcer group and the controls is shown in Table I.

TABLE I.—*Age Distribution*

Age	D.U.s and Controls
15–20	3 (2)
21–25	10 (8)
26–30	14 (12)
31–35	25 (14)
36–40	34 (21)
41–45	54 (34)
46–50	38 (30)
51–55	13 (10)
56–60	27 (14)
61–65	14 (7)
66–70	9 (6)
71–75	5 (4)
76–80	4 (2)
Total	250(164)

The figures in parentheses refer to the 164 cases of duodenal ulcer and their controls from whom fuller details were obtained.

Diagnoses were recorded only for the last 164 patients in the control group (Table II), but there was nothing to suggest that the diagnoses of the first 86 differed in any way from those of this group.

TABLE II.—*Diagnoses of 164 Consecutive Controls*

Surgical		Medical	
Hernias	23	Cardiovascular	11
Appendicitis	15	Neurological	18
Carcinomata	12	Respiratory	14
Genito-urinary	10	Renal	12
Fractures	9	Diabetes	4
Other orthopaedic conditions	10	Miscellaneous	11
Other surgical conditions	15		
	94		70

When the material was collected from the controls, only those giving a history of a definite ulcer demonstrated by *x*-ray film or operation were excluded. Thus the control group may include a small number of unproved ulcers, but none was in hospital for investigation of dyspepsia. No patient, except the above, was excluded once he had given the details required. Approximately 20 patients were found to be incapable of answering the questions owing to their medical state —for example, uraemia, aphasia, and gross senility.

Family Factors

The importance to psychological development of an individual's position in his family, and the size of that family, are well known and are mentioned by numerous authors—for example, Fenichel (1934). The different amount of time that parents with a small or large family can devote to each member of it, the traditional belief that the only child is spoilt, the early assumption of responsibility by the eldest child in a large family, and the tendency of the youngest child, especially if it is a boy, to be regarded as mother's favourite, are all potential factors in the formation of personality. It therefore seemed worth while investigating whether any of these factors could be found to predispose to the development of a duodenal ulcer.

It will be seen from Table III that there is no real difference between the ulcer group and the controls in

TABLE III.—*Number of Siblings in Family*

	Total No. of Siblings, Including Patient				
	1–3	4–6	7–9	10–12	13+
(A) Size of family:					
D.U. (250 cases)	70	96	60	17	7
Controls (250 cases)	75	104	40	24	7

	D.U.	Controls
(B) Position in family:	(250 cases)	(250 cases)
Eldest child	45	54
Youngest child	50	62
Only child	17	12
*"Effective" only child	13	21
*"Effective" eldest child	8	6
*"Effective" youngest child	17	15
(C) Position among male siblings:	(164 cases)	(164 cases)
Eldest boy	40	42
Youngest boy	45	56
Only boy	22	22
(D) Sex of siblings:	(164 cases)	(164 cases)
No. of brothers	328	351
No. of sisters	373	389

* By "effective" is meant a separation from the appropriate sibling or siblings by six or more years.

the number of siblings. The slightly greater number of only children among the duodenal ulcers is not statistically significant. The mean numbers of siblings in the duodenal ulcer group of 250 cases and their controls are 5.6 and 5.5 respectively.

Nor could any evidence be found to support the contention of Ruesch that patients with duodenal ulcer tend to be the younger children. In fact, there were slightly fewer youngest children among the duodenal ulcer group than among the controls. In any unselected community the number of eldest children can be expected to be slightly less than the number of youngest, and this is what was found in both groups.

A comparison was also made between the two groups in the number of patients who were separated from the next sibling, older or younger, by a period of six years or more. It was felt that the position of a boy who had been for many years the only or the youngest child, and then has his position threatened by the arrival of another sibling, was one that might reasonably lead to psychological difficulties. Thus the figures are given for what is termed "effective" only child—that is, one who is separated from the nearest sibling by six or more years—and for "effective" eldest and youngest who have six years between themselves and the next older and younger sibling respectively. This also produced a check on Ruesch's statement that patients with duodenal ulcer tend to be separated from their next older or younger sibling by a space of several years, which isolated them from the rest of the children.

It will be seen that there is no difference in this respect between the groups. In the 164 more fully documented duodenal ulcers and their corresponding controls the position of the patient among the boys of the family was also recorded. Again there is no difference.

The number of brothers and sisters of these 164 were also recorded to check the statement of Draper and Touraine that ulcer families tend to produce more males. It will be seen that for both groups there is a slight excess of females. There is again no difference between the ulcer group and the controls.

Parental Factors

The early loss of a parent, especially the mother, by death or separation is obviously an important factor in character formation. It has been shown that early

bereavement is a statistically significant factor in psychoneurosis and psychosis (Barry, 1949). No difference was found in either the number of patients with duodenal ulcer who had lost a parent before the age of 15, or of those who were separated from their parents before the same age (Table IV). Moreover, there is

TABLE IV.—*Parental Factors*

	D.U.	Controls
When patient 15 or less:	(250 cases)	(250 cases)
Death of father	39	39
,, ,, mother	31	29
,, ,, both parents	5	7
	(164 cases)	(164 cases)
Separation from father	53	50
,, ,, mother	39	46
,, ,, both parents	31	32
,, or divorce of parents	7	14
Remarriage of father	4	9
,, ,, mother	8	10

no evidence here that the traditional part of wicked stepmother (or stepfather) in producing an unhappy childhood is of significance in the aetiology of peptic ulcers, as there were more step-parents among the control group than among the ulcer group. There were rather more divorces and separations of parents among the controls than among the duodenal ulcer group.

The age of 15 was chosen arbitrarily, but the same negative results hold whatever age is taken. The numbers for younger ages are, however, much smaller, and are not given.

Age of Mother.—The mother's age at birth of the patient showed no significant difference between the duodenal ulcers and their controls (Table V).

TABLE V.—*Age of Mother at Birth of Patient*

	D.U.	Controls
	(164)	(164)
Under 30	84	77
30 to 40	62	66
Over 40	8	11
Not known	7	10

Education

It is difficult to assess intelligence without long and elaborate tests, but, in an attempt to find out whether the duodenal ulcer patients showed any greater intellectual ability at school age, details of the 164 duodenal ulcer patients and their controls were obtained (Table VI). It was found that the average child spent one year

TABLE VI.—*Education*

	D.U.	Controls
	(164)	(164)
Secondary school	12	16
Top class more than 1 year	36	38

in the top class of his elementary school, and therefore two rather crude criteria were chosen : (*a*) the number who went to a secondary school (in the population under review this meant in nearly all cases winning a scholarship) ; and (*b*) the number who spent more than one year in the top class.

It will be seen that there is no difference at all between the two groups.

Illness in Childhood

Illness in childhood might possibly affect the development of character in at least two ways : either by leading to an overprotective attitude on the part of the mother, or, if the child were sent to hospital, by developing a sense of insecurity and betrayal by the parents in that it was removed from them just at the time when it most needed parental care—that is, when ill.

Accordingly the 164 patients with duodenal ulcer and their controls were asked whether they had had any illnesses in childhood—that is, at or before the age of 15—other than the usual childish ailments such as measles, whooping-cough, mumps, etc. They were also asked the age at which they had these illnesses, the duration of the illnesses, and whether they were treated at home or in hospital.

The results showed a remarkably close agreement between the two groups (Table VII). The ages at which

TABLE VII.—*Serious Illness in Childhood*

	D.U.	Controls
	(164)	(164)
No. with a history of illness	42	38
No. treated in hospital	20	19

they had been ill and the duration of their illnesses showed very little difference but are not included, as in a large number of cases the patients were unable to give these with any accuracy.

There was little difference in the type of illness—diphtheria, pneumonia, and accidents being responsible for the majority of hospital admissions.

Social Class

Finally, the occupations of the patients in the two groups were compared and also the occupations of the fathers, it being felt that if the man with a duodenal ulcer was "ambitious and hard-driving" he might have risen more in the world than the average, and that by classifying the occupations of both father and son it might be possible to demonstrate such a trend. Moreover, an analysis of the fathers' occupations would be a further check on the childhood background of the two groups, and analysis of the patients' occupations would ensure that the two groups were comparable in this respect as well as in age and the area from which they were drawn.

The occupations were classified according to the five social classes as defined by the Registrar-General (Table VIII).

TABLE VIII.—*Social Class of Father and Patient*

Social Class	Father	Son	Alteration in Class
Duodenal Ulcer Patients (164)			
I and II	18	18	Up 39
III	105	115	Same 87
IV and V	38	31	Down 35
Not known	3	—	
Controls (164)			
I and II	23	13	Up 35
III	100	116	Same 93
IV and V	40	35	Down 35
Not known	1	—	

As might be expected from the population that was being considered, the majority of both parents and

children fell into social class III. For convenience, classes I and II are added together, as their numbers are small, and classes IV and V are similarly joined.

The figures are set out to show a comparison between the social class of the duodenal ulcer group and the controls, and between the fathers of these two groups. In addition, the difference, where there was one, between the social class of father and son has been recorded as "Up" or "Down."

It will be seen that the social class of the duodenal ulcer patients is not significantly different from that of the controls, that the social class of the fathers of both groups likewise does not differ significantly, and that the movement from one social class to another that has taken place has been the same for both groups, the number of those that have raised their social status being approximately equal to those who have declined.

No attempt was made to classify them by actual occupations, as it was felt that the numbers concerned were too small for any conclusions to be drawn, and this has already been done very thoroughly by others.

Discussion

The findings suggest that there is no difference between people with duodenal ulcers and others in the size, composition, or social class of the family into which they were born, in their educational standard at school, or in illness in childhood. Neither do duodenal ulcer patients seem to be more fortunate or less fortunate than others in early separation from parental influence. In none of the points investigated could the findings of other authors be confirmed. It may well be that those points are irrelevant to the development of the "ulcer personality," and that investigation of other childhood features might have shown some important differences between the groups studied: it may be that factors operating in late adolescence or early adult life are responsible for the characteristics said to be shown by these patients. However, unless further investigations show some clear-cut differences between duodenal ulcer patients and the general population regarding factors operating before the appearance of symptoms, the possibility that the "ulcer personality" may be the effect rather than the cause of the disease must be considered.

Summary

In view of the possible aetiological importance of personality in duodenal ulcer a comparison was made between the childhood background of a group of 250 male patients with duodenal ulcer and a similar group of patients with other diseases. No difference could be found between the two groups in any of the points investigated.

I should like to express my gratitude to Dr. F. Avery Jones for his encouragement and inspiration in this work, and to Dr. Richard Doll for much useful advice and criticism.

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AN UNUSUAL CASE OF TRAUMATIC HEART DISEASE WITH CLINICAL RECOVERY

BY

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Cardiac damage, as the result of non-penetrating injuries or blows to the chest wall, has been gaining increasing recognition in the medical literature of recent years. That cardiac lesions of clinical and pathological significance may arise as the result of blows on the chest wall is a fact well established by the work of Barber (1938, 1940), Beck (1935), Warburg (1938, 1940), and others. There has been, however, an unfortunate tendency on the part of certain authors to attribute all sorts of perfectly natural and totally unrelated phenomena of the heart to preceding traumata of insignificant character. Such a tendency can deserve only our severest criticism.

In view of the unusual nature of the cardiac damage and its proved relationship to a previous non-penetrating injury to the chest wall, the following case report is considered worthy of publication.

Case Report

An artillery officer aged 36 has been under our observation since the middle of 1949. Except for childhood ailments like mumps, measles, and scarlet fever, and attacks of malaria and paratyphoid, all within the first decade of life, the patient had enjoyed extraordinarily good health until the time of the accident here described. He had always led an active or athletic life, indulging freely in boxing, swimming, hockey, and cross-country running. There was nothing in the past history to suggest any form of cardiac distress or disease. The family history was also negative, there being no cases of heart disease, sudden death, apoplexy, or high blood pressure.

About the beginning of April, 1948, whilst on a gunnery course at the School of Artillery, he was suddenly struck over the left side of the chest by the "perch handle" of a 25-pounder gun. The sudden impact caused a momentary loss of consciousness, followed by a moderate degree of pain in the chest. During the succeeding weeks he was unable to run or to exert himself on account of the pain: at night the pain used to worry him, necessitating the adoption, throughout the night, of a right lateral decubitus. After a period of three weeks, during which he continued massaging of the chest wall with "iodex," the pain seemed to decrease during the nights, but during the early mornings, when climbing a hill, he experienced a peculiar sense of constriction, which would compel him to stop; this feeling would pass off after a period of nausea and retching.

On two occasions he woke up in the middle of the night with a "strange feeling" in his chest and a profuse "cold sweat"; these feelings were relieved by vomiting. One evening, while cycling, the feeling of constriction in the chest became so intense that he could not get home in spite of all his efforts. A similar sense of constriction in the chest but of much greater intensity overtook him suddenly on the night of May 9, 1948, during a railway journey. The pain was intense enough for him to "scream for assistance." He was carried to a military hospital and given an injection of morphine, in spite of which the pain continued unabated until the night of the 11th. The pain, which was more intense on coughing and deep breathing, for the first time had been bilaterally symmetrical and agonizing,

and was accompanied by a "feeling of deadness" in the left arm and by restlessness, lassitude, and extreme weakness.

On or about June 15 he felt well again and was free of all pain. The systolic blood pressure and erythrocyte sedimentation rate, which had been 104 mm. Hg and 56 mm. respectively in June, 1948, had improved to 124 mm. Hg and 7 mm., respectively, by September of that year. From September, 1948, to August, 1949, there was steady improvement in health and he gradually resumed strenuous duties and arduous hours; there were, however, occasions when sharp and transitory pains were experienced, on and off, in the chest and back.

On August 29, 1949, he again felt a severe pain in the chest, particularly noticeable on coughing and deep breathing and accompanied by fever. He was granted a month's leave and advised to obtain expert advice on his case. An electrocardiogram taken on September 2 (by R. J. V.) showed "atypical right bundle-branch block of Wilson type" with prominent and slurred S waves in leads I, II, and CR_4. The Q wave was prominent in leads CR_1 and CR_2, with inverted T waves. All electrocardiograms taken subsequently have conformed more or less to the same general pattern.

A clinical examination carried out on October 25 was reported as follows:

"A well-built and athletic individual of sober habits and hypersthenic habitus. Teeth good, tongue coated, no engorgement of neck veins, no oedema, and no deformities of chest. The apex beat, neither visible nor palpable, was localized roughly by auscultation as being situated in the fifth left interspace along the mid-clavicular line; this was confirmed by percussion. The second heart sound was found accentuated in both the basal areas, whilst the first sound in the mitral area was both muffled and accompanied by a soft systolic bruit. No pericardial friction sounds were audible at the time. The pulse rate at the time of the clinical examination was recorded as being 54 a minute and the blood-pressure readings as 122 mm. Hg systolic and 76 mm. Hg diastolic. Examination of the other systems revealed nothing of importance except for a small degree of non-tender enlargement of the spleen."

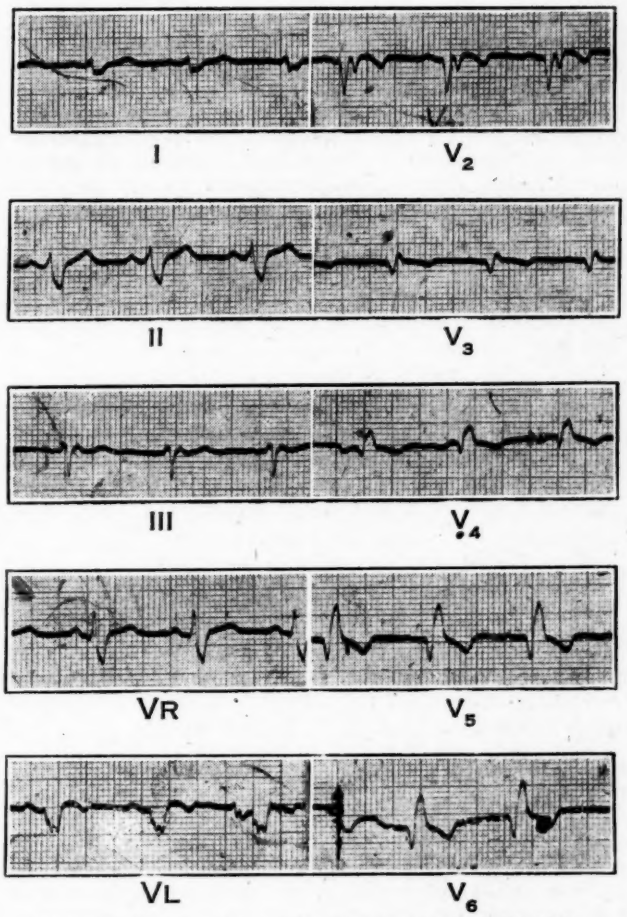
Teleskiagrams were taken, with barium in the oesophagus, on October 6. The radiologist's report was as follows:

"Symmetrical thoracic cage of sthenic type. The straight film (postero-anterior) reveals a well-defined opaque line about 1½ in. (3.8 cm.) long, in the anterior axillary region above the upper borders of the fourth and fifth left ribs, suggestive of periosteal new bone formation. The ribs show normal shape and regular contours. Normal translucency of both lung fields; normal broncho-vascular markings. The cardiac silhouette, in straight P.A. view, shows normal size and shape and smooth outlines; the right lower arch shows normal configuration, and the right cardiophrenic angle appears clear. Normal appearance of the cardiac silhouette in the right anterior oblique position. In the left anterior oblique view the posterior contour of the left lower arch appears somewhat flattened, with a tent-like angulation about one fingerbreadth above the diaphragmatic dome. Also seen in this view is a faint shadow, fairly well defined although less dense than the cardiac shadow, at the cardio-phrenic angle, in front of the right ventricle. The oesophagus, as visualized by barium swallow, shows normal course in all three views."

On fluoroscopy, it was noted that the irregularity at the sinistro-posterior contour of the heart, seen in the left oblique view, was a persistent feature not dependent on the cardiac pulsations. Also, the cloudiness observed in front of the right ventricle was present during both systole and diastole.

On the basis of the above findings it was concluded that a circumscribed pericardial adhesion existed in the region of the posterior aspect of the left ventricle as well as circumscribed mediastino-pericardial adhesions in front of the right ventricle.

The accompanying electrocardiographic tracings, recorded on October 7, 1949, conform closely to one of the varieties of so-called atypical or unusual form of right bundle-branch block, described by Wilson *et al.* (1932, 1934), by Bayley (1934), and by others. In accordance with their criteria, all the ventricular deflections of lead I are of low voltage with the last deflection of QRS directed downwards;



Electrocardiogram of the case taken on October 25, 1949.

also, in leads II and III there is a small R followed by a broad deep S, while T is upright. The QRS duration works out at 0.164 second. The electrocardiographic diagnosis of atypical right bundle-branch block suggested by the standard limb leads is fully substantiated by the precordial leads.

In the present tracing, although the limb leads fail to furnish any evidence of an associated myocardial infarct, the unipolar chest and extremity leads do suggest the coexistence of an anterior infarct. For instance, precordial lead V_2", which faces the epicardial surface of the right ventricle, instead of displaying the typical rsR′ pattern of uncomplicated right bundle-branch block, shows a wide Qrs pattern with an elevated S–T segment and inverted T wave of coronary type. Such a change in pattern, according to Goldberger (1949), is strongly suggestive of an associated anterior infarction. This is substantiated by precordial leads V_3, V_4, V_5, and V_6, which show elevated RS–T segments and inverted T waves in association with QR patterns, facing as they do the epicardial surface of an "injured" left ventricle. Final evidence of anterior infarction is supplied by unipolar extremity lead aVL with its deep and wide QS pattern, elevated RS–T segment, and inverted T wave. The present tracing supports the contention of Rosenbaum *et al.* (1944) that, in the event of a right bundle-branch block and anterior infarction appearing conjointly, it is the precordial lead and not the standard lead of the tracing that clinches the diagnosis.

On October 13 the erythrocyte sedimentation rate at the end of one hour was 6 mm.; the red cell count was 5,250,000 and the leucocyte count 6,500 per c.mm. (59% polymorphs, 38% lymphocytes, 1% monocytes, and 2% eosinophils); and haemoglobin 15 g. per 100 ml. The blood urea worked out at 40 mg., whilst the urea clearance was 59% of average normal.

Since that time we have followed the patient up quite regularly. Although exercise tolerance has improved enormously and he is devoid of all symptoms of heart disease, the physical and electrocardiographic signs remain practically unaltered. Recently, on the suggestion of a colleague, the patient was received in hospital for a few weeks and subjected to a course of anticoagulant therapy with moderate doses of dicoumarol. Though the patient reported some degree of subjective improvement after that treatment, the objective findings remained the same.

Discussion

Experimental proof of cardiac damage or rupture as the result of non-penetrating injuries to the chest wall has been afforded by the studies, in dogs and rabbits, of Külbs (1909, 1937), Schlomka (1934), Kissane *et al.* (1937), and others. They have succeeded in inducing, by the use of controlled and measured traumata to the chest wall, various types of heart lesions, including haemorrhagic and necrotic lesions, cardiac failure, various forms of arrhythmias, and sudden death from ventricular fibrillation and cardiac arrest. Moritz and Atkins (1938) and Bright and Beck (1935) recorded examples of lesions resembling myocardial infarction in animals as the result of blows applied directly to the exposed heart.

Clinical proof of the existence of traumatic heart disease as a true entity is not far behind. In the clinical and pathological reports of Urbach (1922), Kahn and Kahn (1929), Tuohy and Boman (1931), Beck (1935), Barber (1938), and Warburg (1938, 1940), numerous observations are recorded which lend credence to the concept of heart disease arising secondarily to non-penetrating injuries of the chest wall.

The earlier references in the literature to the subject of traumatic heart disease were notoriously pessimistic in their outlook, most of such cases, if not all, being regarded as fatal. For instance, in the series of 175 cases of non-penetrating lesions of the heart reported by Bright and Beck (1935), as many as 163 cases (93%) were reported as proving fatal. Of late, however, it has been increasingly recognized that the percentage of recoveries, either partial or complete, after chest-wall injuries is much higher than has hitherto been believed. The former pessimism has been ascribed by Beck (1935) to "a failure to recognize or a fear to diagnose" traumatic heart disease.

Enough evidence has by now accumulated in the literature for it to be established that a perfectly normal heart with no evidence whatsoever of pre-existing disease (as in the case of the present case) is capable of damage from non-penetrating chest-wall injuries. However, as has been stressed by Külbs and Strauss (1932), Friedberg (1949), and others, a "blunt injury" to the chest wall is more likely to damage a diseased heart than a healthy one. Another fact of prime importance (though not applicable in the present case), recently established by the studies of Bilderbeck (1919), Hamilton (1934), Bright and Beck (1935), Warburg (1938, 1940), and of Reed and Berger (1945), is that serious heart damage, even inclusive of rupture, may ensue in spite of there being no significant or visible external bruising or rib-fracture.

The possibility of a "cause and effect" relationship between a chest-wall trauma and the subsequent development of a myocardial infarct or clinical picture of acute coronary occlusion has been the subject of considerable discussion in medical and medico-legal circles. This subject, obviously of great medico-legal importance, demands a much more extensive and unbiased investigation. For a proper appraisal of this question, however, it is important that we draw a clear line of demarcation between cases of so-called "contusio cordis" or "commotio cordis" (where myocardial haemorrhage with subsequent necrosis and fibrosis arises directly as the result of trauma) and cases of genuine coronary occlusion with myocardial infarction; such a distinction, although theoretically simple, is not easy to determine in actual practice. In spite of an extensive literature on this subject, there are actually not many instances reported of *proved* cases of coronary occlusion secondary to non-penetrating injuries of the chest wall. Because of the possibility of coincidence or an error of interpretation, it is not an easy matter to implicate a previous injury to the chest wall as the causative factor in any given case of coronary occlusion. Schmincke (1925), Warburg (1938, 1940), Kienle (1938), Macdonald (1941), and others have reported cases of this type in which blunt injuries or blows to the chest wall have been directly followed by attacks of typical coronary occlusion. The clinical syndrome of coronary occlusion in these cases has developed either immediately or even days, weeks, or months after the actual causative trauma.

In the case report presented here we find a young and healthy male of athletic habits who, for the first time in his life, develops symptoms and signs suggestive of heart disease from the moment he endures an injury to the chest sufficiently heavy to cause a momentary loss of consciousness. From that time onwards he presents, from time to time, the typical symptoms and signs of acute coronary spasm and, later, of acute coronary occlusion. The diagnosis of acute coronary occlusion is proved by the electrocardiographic picture, the high erythrocyte sedimentation rate, and the pyrexia.

Conclusions

In view of (1) the healthy past history of the patient; (2) the development of anginal symptoms immediately after a severe crush injury of the chest; (3) the radiological demonstration of fairly recent fractures of the fourth and fifth left ribs; (4) the development of definite symptoms and signs of acute coronary occlusion, including an electrocardiographic picture of anterior wall infarction with a typical right bundle-branch block; and (5) the radiological demonstration of pericardial and mediastino-pericardial adhesions in front of the right and behind the left ventricle, we feel justified in regarding this case as one of traumatic or, more correctly, post-traumatic coronary occlusion.

Cardiac arrhythmias of diverse character have been reported from time to time after injuries to the chest wall. Of these, auricular fibrillation is said to be the commonest (Hay and Jones, 1927; Friedberg, 1949. In the opinion of Kahn and Kahn (1929), Levison (1928), Reed and Berger (1945), and others, auricular fibrillation may arise *de novo* in a perfectly normal heart as the result of an injury to the chest wall. It was observed in about a third of Warburg's (1938, 1940) series of 32 cases. Sinus bradycardia has been reported after chest injury by Külbs (1937), auricular flutter by Kahn (1930),

and heart-block, complete or incomplete, by Laubry *et al.* (1921), Gallavardin (1922), Coffen (1930), Tuohy and Boman (1931), and others. But, so far as we can ascertain, this is the first case on record of atypical right bundle-branch block with anterior wall infarction arising as the result of a chest-wall injury and attended by clinical recovery.

Summary

A case report is presented of anterior wall infarction with atypical right bundle-branch block, arising directly after a crush injury to the chest wall, in a perfectly healthy young man and attended by clinical recovery.

So far as can be ascertained, this is the first case of its kind in the medical literature.

A short review is presented of present-day views on the subject of traumatic heart disease.

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Professor A. C. W. Hutchinson, giving his inaugural lecture at Edinburgh University (*Scotsman*, October 30), traced the course of dental history in Edinburgh. In 1876 the Edinburgh Dental Hospital and School was established, owing its beginning to the generosity of a few members of the dental profession who not only provided the funds but also gave up their time to teach the students. The first woman student was admitted 14 years later, and became the first woman to qualify as a dental surgeon; she was Lilian Lindsay, who celebrated her 80th birthday this summer. To-day there were 21 full-time posts on the establishment of the hospital and school, and the major problems were those of demolishing the old parts of the buildings and erecting new ones in their place, and of improving still more the standard of the teaching. Before the war negotiations were begun with the University with a view to making the dental school an integral part of the University. These had to be discontinued during the war, and were resumed after it; in 1948, at the beginning of the Health Service, the school was transferred to the University. The course for the degree of L.D.S. was instituted in 1949. He said the greatest task of all must be to substitute prevention for repair, and as a foundation students should be instructed in developmental factors, including nutrition, and more attention would have to be paid to children's teeth.

DIATHERMY TREATMENT OF ANGIOMATA

BY

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Benign tumours of blood and lymph vessels are relatively common, but there is no unanimity of opinion regarding their treatment. The present-day method of treating angiomata depends on the worker and his tools, and, on the whole, it may be said that the surgeon uses the scalpel or diathermy, the radiotherapist *x* rays or radium, and the dermatologist carbon dioxide snow. As the majority of these tumours are usually located in conspicuous areas, a good cosmetic result is an important consideration.

This paper is concerned with an attempt to improve the diathermy method, which has fallen into disrepute on account of disfigurement and scarring resulting from necrosis and ulceration of the healthy skin through which the current must pass. Nevertheless, in diathermy we do possess a safe and sure agent for the treatment of angiomata, provided that the high-frequency current is applied to the tumour without damage to the skin or the normal tissues through which the electrode is introduced. With this in view a special insulated needle electrode has been prepared and used with great success in more than 80 cases.

Some General Remarks

An angioma is a tumour composed of blood or lymph vessels. Normal blood vessels are derived from a vascular network, and it is said that the primary factor in the development of an angioma is the persistence of part of this network. They are therefore not true neoplasms but anomalies of development. Clinically the angiomata are divided into two distinct groups: (*a*) the haemangioma, which is composed of blood vessels containing circulating blood; and (*b*) the lymphangioma, which consists of lymphatic vessels and contains lymph. The subdivisions of these two groups which are suitable for treatment are discussed below.

It has been stated (Ribbert, 1898) that angiomata have no anastomotic communications with surrounding blood vessels, but my experience does not confirm this view. If the blood supply were a limited one, many of the attempts to destroy this tenuous supply would cause spontaneous disappearance. Cure cannot be expected unless every portion of an angioma is destroyed.

The smaller haemangiomata seen in children often disappear without treatment, but spontaneous disappearance of the larger lesions should not be awaited, because early treatment with low-intensity diathermy is safe and gives the best result.

Method

The diathermy coagulates the tissues by an intense heat generated in the vicinity of the active electrode. If the needle electrode is non-insulated the tissues surrounding it will be charred throughout the length of the needle. This causes unnecessary injury to normal tissues. In the method about to be described the needle is insulated except at its tip, and in this way the current is delivered only to the spot which requires treatment, and damage to the normal structures is avoided.

Preparation of the Special Needle

Straight intestinal needles of various sizes and lengths are generally chosen, but curved ones may also be used. All grease is removed from them by washing in ether, and the blunt end of each is then pushed half an inch into a cork. The cork acts as a holder for the needles, which are then ready for insulating. This is done by immersion in a jar of wire enamel* which is similar to that used for the insulation of high-tension electric cables, the ends of the needles embedded in the cork remaining non-insulated. The needles are withdrawn, allowed to drain for a few seconds, and then placed upright in an oven and baked at 180° C. for 15 minutes. This time should be sufficient to harden and dry the enamel. Lower temperatures will take longer. The immersion is repeated twelve or more times until it is certain that the insulating coat is thick enough to resist the diathermy current. When the needles are sufficiently insulated they acquire a deep mahogany colour. On completion of the baking process they are removed from the cork and 1–2 mm. of the insulation is removed from the pointed ends with a scalpel blade. They are then placed in a test-tube and autoclaved before use.

The choice of a long or short, straight or curved needle depends on the site and size of the angioma. The uninsulated base of the needle is clamped in the groove in the jaws of a diathermy haemostat (Aiken, 1944) or fixed in the usual bakelite holder and connected to the diathermy machine. Either the spark-gap or valve diathermy may be used, but the former is more satisfactory.

Anaesthetic

As a rule I use intravenous thiopentone for adults and ethyl chloride or "trilene" for infants. Ethyl chloride may be used with safety, because the coagulation is carried out in the depth of the tissues and there is no external sparking to cause an explosion.

Skin Preparation and Site of Puncture

The majority of patients attend as out-patients and the skin is prepared with spirit before administration of the anaesthetic. The needle, lubricated with petroleum jelly, is introduced through the skin or mucous membrane at some little distance from the tumour and pushed along through the normal tissues into the tumour. A cavernous angioma of the tongue may be treated by inserting the needle through the skin of the submental triangle. This technique has the advantage that the insulated needle delivers the current to the tumour without burning the normal skin or other normal tissues adjacent to it, and the brisk bleeding which follows direct puncture of the lesion is avoided.

Application of the Current

A small tumour will need the application of the current at one point only, whereas a large angioma should be treated at multiple points—1–2 cm. apart—and it is better to treat a segment of it rather than attempt its complete destruction at one session. The current must never be of high intensity, and it is most important that it should be applied only for a few moments, say two to five seconds at each point. With longer applications the great heat generated is dissipated throughout the tissues, and if it is too intense the skin will slough. The surface heat should be just perceptible to the operator's bare finger, which acts as an additional guard against overheating. If overheating has occurred, spread to the skin must be prevented by cooling the area with a swab soaked in cold water. Furthermore, the point of the needle should never be allowed to come too near to the surface during the treatment, because sloughing of the surface tissue may take place and compromise the result. This risk is easily avoided by careful palpation of the needle point before turning on the diathermy current. A semi-darkened room is an advantage, as the radiating sparks give an indication of the proximity of the needle point to the surface.

Those not familiar with the method are recommended to treat the lesions frequently with a low-intensity current rather than to risk overheating the part. In order to obtain the best results it is a wise principle at all times to undertreat rather than overtreat, aiming at resolution with the minimum of scar formation.

*The wire enamel was kindly supplied to me by Messrs. Connolly, of Blakley, Manchester.

After-treatment

The part becomes swollen within a few hours of treatment, but, except in young children, no dressing is required unless the skin is blistered. Should this happen "tulle gras," covered with dry gauze, may be applied and held in position with strapping or a bandage. In infants it is always wise to cover the treated lesion to prevent scratching and subsequent infection. Angiomata in the mouth do not require any after-treatment other than the usual attention to oral hygiene. Mouth washes of glycothymoline are recommended. Repeated sessions may be necessary to complete the treatment, and these are best carried out at intervals of three to six weeks until the entire tumour has disappeared. Large and difficult tumours need careful follow-up, and recurrences should be treated as they arise. The patient suffers little inconvenience.

Results

Over 80 angiomata have been treated in the past five years with the special needle here described. They include the following types.

Blood and Lymph-vessel Tumours

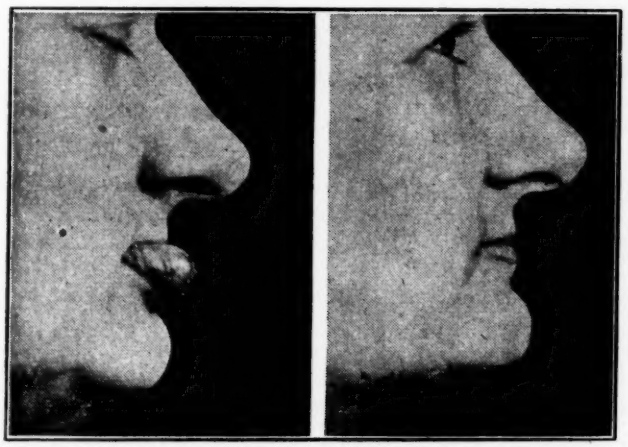
Capillary angioma	45
Cavernous angioma	32
Racemose type	1
Port-wine stain	1
	79
Capillary lymphangioma	2
Cystic hygroma (cavernous)	1
	82

Treatment is usually sought for cosmetic reasons, and the ultimate prognosis depends on several factors. Generally, it may be said that the larger the original tumour the greater will be the disfigurement, and this applies particularly to the capillary lesions, which are so superficial. Even in these, however, when treatment has been completed, the epithelium becomes thicker and the mark less noticeable as the years go by. Any difference in the appearance can be skilfully covered by judicious cosmetics. It should be remembered that all forms of treatment entail a certain degree of scarring.

Capillary Haemangiomata.—These formed the largest single group treated by the special needle. The small growths were all readily and easily treated. Those of several centimetres diameter were tedious to treat, but the response was always good and there were none of

the untoward sequelae from diathermy, such as interference with the epiphysial growth, damage to the eyes, or epilation, that may result from radiotherapy. The superficial nature and delicate endothelium of the large capillary angioma tempted me to treat a few with a flat-button electrode of 7 mm. diameter. Cosmetic results were excellent, and the method was less tiring than treatment from beneath by the insulated needle, but a low-intensity current must be used to avoid overheating the tissues, with subsequent sloughing and scar formation. The latter method is undergoing further trial with electrodes of various diameters.

Cavernous Angiomata.—These are generally situated deeply and are covered by healthy skin or mucosa, little or no blemish being left after treatment. Some of my best results were obtained in these cases (see illustration),



Photographs showing cavernous angioma of lower lip before and after treatment with the insulated needle.

the response often being dramatic. With care the tissues and configuration of the area—apart from the deep scarring in the tumour—may be returned to normal. Some of the cases had been treated by irradiation previously, and in these diathermy was used carefully because of the danger of sloughing of the irradiated tissue.

Port-wine Stains.—These present a problem best left to the plastic surgeon. In the one case treated there were obvious capillary overgrowths in parts of the stain. The recorded case responded well and the patient was delighted with the result, but the treatment was an arduous and difficult undertaking. The lesion involved the right side of the face and both eyelids, and great care was needed to avoid scarring and resultant contractures. The port-wine portion of the stain remained almost entirely unaffected by the treatment.

The two *capillary lymphangiomata* occurred in the tongue. They were considerably reduced in size and showed marked improvement after four treatments with the needle electrode. Neither case has required further attention for the past two years.

The *cystic hygroma* was a small one situated in the left supraclavicular fossa. It disappeared completely after a single treatment by the needle electrode four years ago, and there has been no recurrence. Because of the remarkable power of growth which these tumours possess, treatment at the onset is advocated.

In the present series there was only one case of *cirsoid aneurysm*, and it involved the left superficial temporal artery. The main artery was ligated and the remaining secondary varicosities were treated with the diathermy needle electrode on four occasions. The tumour disappeared entirely, and when the patient was examined two years later it had not recurred.

I have had no experience of the treatment of the angiomata involving a whole limb, and I very much doubt if this method of treatment could be applied with success. Fortunately these extensive lesions are rare.

Summary

A series of 82 patients with angiomata have been treated with diathermy, and these have been followed up for one year after treatment. All forms of angiomata, except the port-wine stain and angiomata involving a whole limb, may be treated with a specially insulated needle. This method is simple and safe, and gives a cosmetic result which is comparable with that of other methods of treating capillary angiomata, and is superior to them in the treatment of cavernous angiomata. The preparation of the special needle and the way to use it are described.

I wish to thank Mr. Wilfred Hynes, our plastic surgeon, for his great help and criticisms, and for allowing me to treat patients under his care. I am also grateful to Mr. H. Blacow Yates and Dr. E. R. A. Cooper for the help they have given me.

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USE AND INTERPRETATION OF THE FANTUS ESTIMATION OF URINARY CHLORIDE

BY

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Since its advocacy by Marriott (1947), the ward test of Fantus (1936) for estimating urinary chloride ion concentration has been widely used in clinical practice. This communication describes experiments which have been made to determine its reliability, and records circumstances in which interpretation of the test in the normal fashion would lead apparently to mistaken conclusions about the electrolyte requirements of certain patients.

Methods

Fantus Test.—The method described by Fantus (1936) was used. It is essentially a modification of the Mohr (1856) titration. To 10 drops of urine a drop of 20% potassium chromate solution is added. With the same dropper, silver nitrate solution (29 g. per litre) is added drop by drop until a permanent and distinct colour change to red-brown occurs. The number of drops thus required is taken as a measure of the content of chloride in grammes of NaCl per litre.

Volhard Titration.—The method described by Peters and Van Slyke (1932) was used.

Results: (a) Anions Estimated by the Test

It is usual to estimate chloride ions in urine by a method involving their precipitation as silver chloride in acid solution, as in neutral or alkaline solution other silver compounds may be precipitated and render the

estimation inaccurate. The Fantus test is, of course, carried out in neutral solution, or nearly so, depending upon the *p*H of the urine. The extent to which the formation of other compounds renders the test unreliable must therefore be determined. This was done by the addition of various anions in the form of the solid sodium or ammonium salts to 10 drops of urine and then performing the test.

Of the anions commonly present in urine, sulphate, phosphate, and bicarbonate were without effect upon the result of the test. Urates, however, did affect the result, and the magnitude of this effect must be assessed. The normal daily excretion of urates in urine is 0.1–2 g. (Harrison, 1947) measured as uric acid, and the normal urinary volume is 1–1.5 litres. The normal concentration of uric acid in urine could therefore vary from 0.007 g./100 ml. to 0.2 g./100 ml. Uric acid is known to form monobasic and dibasic salts with cations, and, assuming that one molecule of silver nitrate reacts with one of uric acid, then one drop of the 2.9 g./100 ml. silver nitrate solution used in the Fantus test is equivalent to one drop of 2.8 g./litre uric acid solution, or to 10 drops of a 0.28 g./litre solution. At the most, therefore, the amount of urate present in 10 drops of urine used in the Fantus test would utilize 0.7 drop of the silver nitrate solution. If the di-silver urate were wholly formed, 1.4 drops would be utilized.

Carbonate ion was also found to interfere with the test. The amounts normally present in urine are small, but in urine which has stood for some time, especially if infected, urea is converted into ammonium carbonate, and this may cause an appreciable error. It was found that the Fantus test result may be increased by up to 2 g./litre if the urine is allowed to stand for 24 hours at room temperature. The presence of silver carbonate in the reaction mixture at the end-point of the Fantus test can often be demonstrated by the addition of a mineral acid to the filtered precipitate. There is an effervescence of carbon dioxide, which can be detected by the usual tests. It is important, therefore, always to perform the test upon a fresh urine specimen.

There are other factors which could potentially affect the result of the Fantus test. The chromate ion is only stable in the *p*H range 6.5–9 (Vogel, 1939). In more acid solutions the reaction

$$2CrO_4^{--} + 2H^+ = 2HCrO_4^- = Cr_2O_7^{--} + H_2O$$

occurs. The tendency of this would be to reduce the chromate ion concentration and delay the appearance of the end-point in an acid urine. A further factor which may be of significance in a urine containing large amounts of ammonium ion—that is, when infected or in acidosis—is the ability of silver ions to form the argentammonium ion $(Ag(NH_3)_2)^+$. This factor would again tend to increase the number of drops of silver nitrate solution used.

(b) Error in Performance of the Test

The accuracy of the test depends also upon producing drops of equal volume from a dropping pipette with rubber teat. The ability to do this depends in turn on the experience of the observer. Thus, two biochemists and three technicians who were accustomed to using dropping pipettes made many estimations on the same urine specimen, giving results shown in Table I.

Three house officers and two medical students, unaccustomed to using the method, gave a rather wider scatter (Table II).

TABLE I

Correct Urinary Chloride Concentration (g./litre)	No. of Drops of $AgNO_3$ Used	No. of Readings
1·9	2	10
	3	2
8·5	8	3
	9	9
4·0	7	4
	8	8
	9	1
4·5	4	1
	5	7
	6	1
4·5	4	2
	5	6
	6	1

TABLE II

Correct Urinary Chloride Concentration (g./litre)	No. of Drops of $AgNO_3$ Used	No. of Readings
8·5	6	2
	7	2
	8	5
	9	2
	10	1
5·7	5	2
	6	1
	7	7
	8	2
6·2	6	1
	7	4
	8	13
	9	4
4·0	6	1
	7	6
	8	3
4·0	5	2
	6	5
	7	2
	8	1

These results afford an illustration of the inability of the test to give reproducible results even in the hands of the more experienced manipulators, and the decreased reliability with those less experienced. The urine containing 4 g. NaCl/litre was markedly alkaline and contained much carbonate ion, giving high Fantus test values which illustrate well the points made in the preceding section.

It should be noted, however, that the smaller the amount of chloride ion in the urine the less will be the inaccuracy caused by drops of unequal size, as fewer drops will be needed.

(c) Effect of Errors in Practical Use of Test

In 62 urine specimens the results obtained by the Fantus test were checked by the Volhard titration. The Fantus test was in each case performed by the same observer on urines which had stood for not longer than four hours, so that errors due to the formation of ammonium carbonate and to inexperience with a dropping pipette were minimal. The results are given in Table III.

TABLE III

Volhard Titration g./litre	No. of Urines Tested	No. of Fantus Test Results Giving Error of	
		1–2 g./litre	2–3 g./litre
0–1	26	8	2
1–2	5	0	0
2–3	3	1	0
3–4	8	4	0
4–5	3	0	1
5–6	5	2	0
6–7	5	3	1
7–8	2	1	0
8–9	1	0	0
9–10	3	1	1
10–11	1	1	0

The largest error is 2–3 g./litre, and this occurred in 5 out of 62 estimations (8%). An error of 1 g./litre or over occurred in 26 out of 62 estimations (42%).

From a statistical point of view these specimens show a preponderance of urines containing little chloride ion. The natural accuracy of the test is greater in these urines, so that the results expressed show the test in as favourable a light as possible.

(d) Interpretation of the Results of the Fantus Test

Marriott (1947) considers that "in concentrated urine, with a specific gravity of more than 1020, less than 3 g./litre (of chloride measured as NaCl) suggests (salt) depletion." Black (1950) considers that a urinary salt concentration of less than 5 g./litre indicates the need for increasing salt intake. He makes the reservations that the urine must be reasonably concentrated, that there must be no disease of the suprarenal glands, and that the Fantus test may fail to show salt deficiency in the presence of acidosis. Marriott also mentions that during intravenous saline therapy there may be an adequate concentration of chloride ion in the urine although the body is still salt-deficient.

There are patients, however, in whom such an interpretation of the Fantus test may be misleading: first, those in whom there is hyperchloraemia and hypernatraemia but a urinary chloride ion concentration of less than 1 g./litre in a concentrated specimen; and, secondly, those with hypochloraemia and hyponatraemia yet with a high urinary chloride concentration and symptoms of salt deficiency but with no evidence of Addison's disease. This second group may be subdivided into those (mentioned by Marriott) in whom saline transfusion is being carried out and those in whom there is no such therapy.

During the past year 12 patients of the hyperchloraemia group have been noted in the Radcliffe Infirmary. They consist of 6 fatal cases of head injury, 1 non-fatal case of perforated peptic ulcer (post-operative), 1 fatal case of carcinoma of the oesophagus (post-oesophagectomy), 1 fatal case of fat embolism (post-traumatic), 1 fatal case of diabetic coma, 1 fatal case of meningitis and lung abscess, and 1 fatal case of post-operative partial gastrectomy. The cases with head injury have been reported in some detail in a separate communication with other workers (Higgins *et al.*, 1951).

The biochemical findings in the following patient are fairly typical.

Case Report

A man aged 57 with a 20-year history of epigastric pain characteristic of peptic ulceration developed the symptoms of pyloric stenosis. Seven months previously he had had an attack of renal colic, but a subsequent intravenous pyelogram had shown good concentration of the dye and no calculi in the kidneys. The blood-urea concentration was 33 mg./100 ml. After a period of preparatory medical treatment, a Billroth I partial gastrectomy was performed. Post-operative progress was stormy, with recurrent haematemesis and a persistently low blood pressure. Two weeks after the operation a hypostatic chest infection with rise of temperature developed, and the patient died.

For the first two days after operation fluid balance was maintained mainly by intravenous transfusion: a total of 1,140 ml. (2 pints) of blood, 1,140 ml. (2 pints) of 0.9 g./100 ml. saline, and 1,700 ml. (3 pints) of Ringer–Locke solution was given. By mouth, 1,140 ml. of water was given. The measurable urinary output totalled 1,730 ml. (61 oz.) and the stomach-tube aspirations 1,250 ml. (44 oz.).

Thereafter for five days an average oral daily intake of 2,850 ml. (5 pints) of water or 50% milk in water was given. Aspirations averaged 1,140 ml. (40 oz.) daily, and collectable urine 910 ml. (32 oz.) daily. During the last three of these days a total of 3,410 ml. (6 pints) of 5% glucose solution and 1,140 (2 pints) of 0.9 g./100 ml. saline was given.

At this stage, on the seventh day after operation, the plasma chloride ion concentration was 700 mg. NaCl/100 ml. (or 120 mEq/litre) and blood urea 176 mg./100 ml. The urine, of specific gravity 1015, was completely free of the chloride ion. Treatment was designed to reduce this high chloride level. Over the next four days an average oral intake of 2,840 ml. of milk, water, and glucose was given with 1,700 ml. of 5% glucose solution intravenously daily. The only sodium chloride received was in the milk. The aspirations averaged 1,360 ml. (48 oz.) daily and the measurable urine 1,140 ml. (40 oz.). The plasma chloride levels fluctuated as follows: 690, 700, 790, and 650 mg./100 ml. (118, 120, 135, and 111 mEq/litre) over the next four days. The serum sodium level on the last of these days was 162 mEq/litre (372 mg./100 ml.) and the plasma bicarbonate 15 mEq/litre (35 vols./100 ml.). The blood urea, after rising to 417 mg./100 ml., fell to 271 mg./100 ml. Throughout this time the urinary chloride concentration varied between 0 and 0.4 g./litre, as measured by the Volhard method. The urinary urea concentration on two occasions was 2.6 and 3 g./litre. Fantus test estimations made by the nursing staff usually gave urinary chloride concentrations of 0–1 g./litre and 1–2 g./litre, although on two occasions misleadingly high values were obtained—2–3 g./litre and 3–4 g./litre.

At this point, 11 days after the operation and five before death, chloride ion was passed for the first time and the urinary concentration rose to 4.5 g./litre. However, chest infection developed and the patient became febrile and generally more ill. It proved impossible to give more than 1,700 ml. (3 pints) of fluid by mouth daily. Blood, glucose solution, hypotonic saline, and sodium sulphate were given intravenously in addition. The patient's condition deteriorated steadily and he died 16 days after the operation.

At necropsy there was thrombosis of several mesenteric veins, causing gangrene and perforation of the colon at a point near the recto-sigmoid junction. Uraemic ulceration of the large and small bowel and bilateral pulmonary congestion and oedema were also found. The coronary arteries were atheromatous, with narrowing of the lumina. There was no significant renal disease, macroscopically or microscopically.

Comment

Had this patient been given intravenous saline therapy during the period of hyperchloraemia and hypernatraemia, as the urinary chloride tests would have indicated, it seems likely that the plasma sodium and chloride levels would have risen even higher.

During the same period 10 patients falling into the second, or hypochloraemic, group were noted. Six were receiving intravenous saline transfusions post-operatively, and four not. Three of the six were children. One, a boy of 11 years subjected to investigative laparotomy, passed 7.5 g. NaCl/litre in a urine of specific gravity 1012 when his plasma chloride level was 87 mEq/litre (510 mg./100 ml.) and his serum sodium level 128 mEq/litre (297 mg./100 ml.). Another child of 8 weeks, following Ramstedt's operation for pyloric stenosis, passed 5.4 g. NaCl/litre in his urine on a day when his plasma chloride level was 82 mEq/litre (480 mg./100 ml.) and he appeared clinically to be salt-deficient. Of the three adults, two were post-abdominal operation cases and one was in diabetic hyperglycaemic coma.

The remaining four all suffered head injuries severe enough to keep them unconscious for several days.

Despite the maintenance of an adequate milk, water, and glucose intake, and an adequate urinary output, they developed unmistakable signs of salt deficiency. One man aged 84, for example, was thought to have had a cerebral thrombosis causing him to fall and sustain a head injury. Five days later the normal skin turgor was lost, the intraocular tension was diminished, the cheeks were hollowed and sunken, and the tongue was dry and wrinkled. His urine was found to contain 7.8 g. NaCl/litre and his serum sodium ion concentration was 130 mEq/litre (300 mg./100 ml.) and plasma chloride ion concentration 82 mEq/litre (480 mg./100 ml.). No intravenous fluids had been given. These four patients responded slowly to Ringer–Locke solution by mouth. Had the Fantus test been performed and interpreted in the usual way, this fluid would have been denied them, as they would not have been considered salt-deficient.

Discussion

The foregoing results pose two separate problems : first, the accuracy of the Fantus test as a measure of urinary chloride ion concentration ; and, secondly, the value of that concentration as a guide to sodium and chloride ion requirements in disease.

Assuming for the time being that the urinary chloride concentration is of some value, it is important that the Fantus test should give a fairly accurate result for values from 0 to 5 g. NaCl/litre, over which range it is going to provide a basis for Na and Cl therapy. Although, for reasons given above, the natural accuracy of the test is greater at lower urinary chloride concentrations, the values obtained under conditions approximating to the best still show an undue amount of error. A urine containing less than 1 g./litre ought not to be estimated as containing 2–3 g./litre as it was in 2 out of 26 cases. In the hands of less practised persons, such as house officers and nursing staff, an error of this magnitude might occur more frequently. In the case described, for example, such urines were twice estimated as containing 2–3 g./litre and once as containing 3–4 g./litre. It is surely indefensible that such results be used as a basis of treatment, especially when the Volhard estimation is in itself so easy and accurate and involves the use of only two solutions, a pipette, a burette, and a conical flask.

The usefulness of urinary chloride estimations may be questioned on a number of grounds. In the first place, sodium is probably the most important ion maintaining extracellular fluid volumes, and urinary chloride ion variations are of use only in so far as they reflect changes in urinary sodium ion concentration. This they may fail to do in acid (Black) or alkaline urines, for example.

Secondly, their usefulness is considerably diminished if they do not reflect the need of the body for sodium ions. The schemes of treatment given by Marriott and by Black assume that they do reflect this need, with the exceptions stated. There is considerable evidence to support this, particularly in the salt-depleted states produced in the Tropics (Marriott) or by forced sweating (McCance, 1936) in otherwise normal individuals. In most patients in whom problems of fluid balance arise in hospital this relationship between urinary chloride concentration and the body's requirement of sodium ions still holds good, but exceptions arise. Those quoted in this paper have all come to light in a hospital of 842 beds in one year. The hyperchloraemic group is discussed more fully elsewhere, where it is argued that the primary disturbance is inability of the kidneys to excrete the chloride ion. An intravenous saline transfusion or oral salt intake will then produce high plasma chloride and sodium levels. Use of the urinary chloride concentration as a guide to therapy would lead to precisely this state of affairs and run the risk of oedema, particularly of the lung bases.

The hypochloraemic group of patients, who pass a urine of relatively high chloride concentration while receiving saline transfusion, are most interesting and need characterizing further. Again, only the minority of patients so treated behave in this way.

The remaining group of patients lose both sodium and chloride ions spontaneously and become progressively salt-deficient. They behave as though they had Addison's disease, and appear to differ essentially from the transfusion group, who may not pass chloride ions in the urine prior to transfusion. Again, it is apparent that use of the urinary chloride concentration would lead to treatment quite the reverse of that required.

Other workers have recently criticized the value of urinary chloride tests. Wilkinson *et al.* (1950), after investigating sodium and chloride balance post-operatively, remark : " It is a corollary of the hypothesis we have advanced that determinations of urinary chloride afford no reliable guide to the requirements of salt and water during the immediate post-operative period, excellent though they are in other circumstances." Spencer (1950) similarly remarks : " Chloride determinations on the urine are sometimes valuable guides to diagnosis and treatment, but they do not necessarily reflect the balance of chloride and sodium in the body." He then goes on to quote examples from the literature.

The conclusions seem to be (*a*) not to use the Fantus test if a more accurate method is available, and (*b*) never to rely on a urinary chloride estimation alone as a basis for salt therapy, but to take into account also the clinical signs and the plasma chloride (or, better, sodium) concentration.

Summary

The errors and reliability of the Fantus test have been investigated.

Its use may lead to serious error in urinary chloride estimations, especially in the hands of inexperienced persons.

The urinary chloride concentration may not be an accurate guide to salt therapy. Examples where this is the case are given.

I wish to thank Dr. V. Smallpeice, Professor L. J. Witts, Mr. A. Elliot Smith, and Mr. J. C. Scott for permission to use the case records of patients under their care, and the staff of the Biochemistry Department for performing the blood analyses in the case described. My thanks are also due to the technical staff, house officers, and medical students who performed the experiments mentioned in Section B, and to Mr. J. R. P. O'Brien for advice and encouragement.

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THE CONCENTRATION OF OXYGEN REACHED IN OXYGEN TENTS

BY

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Oxygen tents are now almost universally used in hospital nurseries, yet there seem to be few published figures giving the concentration of oxygen reached. The experiments described below have been done to determine these concentrations under controlled and reproducible conditions, such as are likely to be found in such nurseries. A standard Queen Charlotte type oxygen tent has been used throughout. The results obviously apply only to this type of tent, but as many nurseries use it the results may be widely applicable. The concentration of oxygen reached with different rates of flow, with the lid closed or open and with or without preliminary flushing of the tent with oxygen, has been estimated. In order to collect data for a number of different rates of flow it was decided to use a rabbit as experimental animal rather than try to collect samples from tents in the maternity department. Results obtained from tents in the hospital closely parallel those arrived at with the experimental tent.

Methods

The tent rested directly on a cot mattress covered with mackintosh, no special precautions being taken to avoid gas leakage. During the experiment an adult rabbit weighing 5 lb. 9 oz. (2.5 kg.) was kept in the tent to mix the contents by its movement and to reproduce the gaseous exchanges occurring in a tent containing a child. It was felt that the oxygen consumption and carbon dioxide production of such a rabbit would be greater than that of the average small baby.

The oxygen was introduced into the tent from an oxygen cylinder which had a flow-meter attached, and the cylinder was connected to the metal inflow pipe on the tent by means of a stout rubber tube. From a point as nearly as possible at the middle of the tent, samples were taken at intervals through a sampling port provided in the side wall, using a piece of glass capillary tube connected to rubber bellows. The samples were collected in rubber balloons and analysed in a standard Haldane gas-analysis apparatus, the accuracy of which was checked by the analysis of known gas mixtures.

The longest period for which a tent is likely to remain closed in clinical use is three hours, and in most instances sampling was stopped at that time, although with the higher rates of flow the oxygen concentration was still rising. Some experiments were continued longer in order to follow the progress of the changes.

Results

The results are expressed as graphs which show the oxygen concentrations reached within the tent under various conditions. Fig. 1 shows that, with the tent shut and without any preliminary flushing with oxygen, a flow of 1 litre of oxygen a minute through the tent gives a concentration of 70% oxygen after three hours. A flow of 3 litres a minute reaches 75% oxygen in one hour and 84% after three hours. A flow of 4 litres a minute gives the very high concentration of 87% oxygen at one hour.

Fig. 2 shows the effect of a preliminary flushing with 6 litres of oxygen a minute for five minutes (a common practice in hospitals) on the concentrations reached with a flow of 1 litre a minute. Comparing Fig. 2 with curve A of Fig. 1, we see that flushing causes a rapid rise to about 75–80% oxygen in the tent, which is maintained so long as the tent is shut. A permanent increase from 70% to 80% is therefore produced by this preliminary flushing.

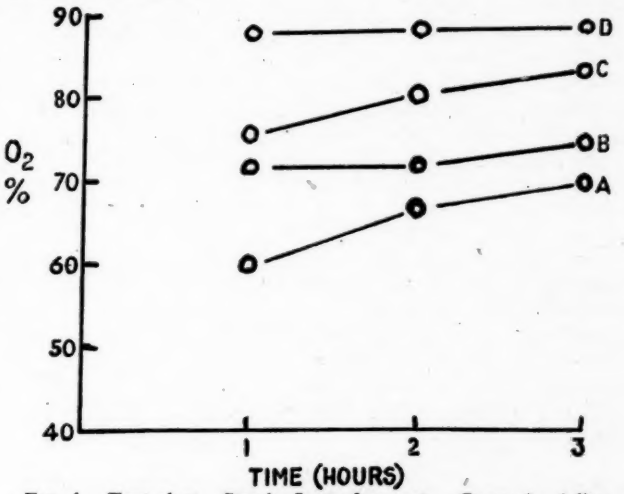

FIG. 1.—Tent shut. Steady flow of oxygen. Curve A=1 litre a minute. Curve B=2 litres a minute. Curve C =3 litres a minute. Curve D=4 litres a minute.

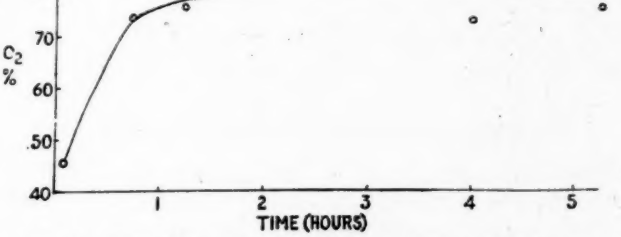

FIG. 2.—Tent shut. Oxygen flow at 6 litres a minute for 5 minutes, then maintained at 1 litre a minute (composite curve from three separate experiments).

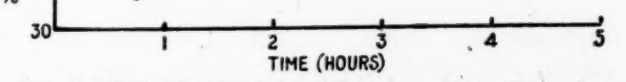

FIG. 3.—Tent lid open 1 in. (2.5 cm.) at foot. Curve A= oxygen flow at 2 litres a minute. Curve B=oxygen flow at 6 litres a minute for 5 minutes, then maintained at 2 litres a minute.

Fig. 3 shows the effect of keeping the tent lid open 1 in. (2.5 cm.). This was done because it is not uncommon for tents to be used clinically in this way. In this case a flow rate of 2 litres of oxygen a minute gives a permanent concentration of 40–45% oxygen in the tent. Comparison with curve B of Fig. 1 shows that if the tent is shut a flow of 2 litres a minute gives a concentration of 70–75% oxygen in the tent. Therefore opening the lid 1 in. (2.5 cm.) almost halves the oxygen concentration at this rate of flow.

The concentrations of carbon dioxide are not shown on the graphs but were often estimated. Only once did the concentration of CO_2 in the tent rise above 0.1%—when the animal was particularly active and the CO_2 concentration rose to 0.2%.

These results can be compared with those of Mackenzie (1950), who, though mainly concerned with CO_2 levels in tents in clinical use, also estimated oxygen in a small number of cases. Though he made no estimations beyond the end of the first hour, the figures given for oxygen are similar to those reported here. There are considerable discrepancies between concentrations of CO_2 found in the tents in the two series of experiments, but this may be due to differences in the weight and general condition of the occupant. A large series of estimations of the gas concentrations in tents in clinical use is needed, as Mackenzie's figures for CO_2 are much higher than those I found in the relatively small number of tents examined in hospital.

Summary

The oxygen concentrations reached in a standard oxygen tent under controlled conditions and with varying rates of flow have been estimated.

An adult rabbit was used as experimental animal.

It would appear that high concentrations can readily be achieved with low rates of flow.

The CO_2 concentration rose above 0.1% on only one occasion.

I am grateful to Oxygenaire Ltd. for the loan of the oxygen tent used in these experiments.

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Medical Memoranda

Rupture of Rectus Abdominis Muscle

Spontaneous rupture of the rectus abdominis muscle is a comparatively rare occurrence; therefore it is worth while to record four cases that have occurred in the practice of this hospital during the past five years. These are all the more unusual as each was quite independent of pregnancy.

The rarity of this catastrophe is such that Wahlgemuth could find only 127 cases in the literature up to 1923, and, later, Torpin (1943) collected 27 cases since 1900, but all these had occurred during pregnancy.

The aetiology of the condition is somewhat obscure, but it is well known that febrile diseases may be followed by muscle degeneration, and that with slight trauma superimposed this may lead to rupture of the muscle. Typhoid fever, influenza, and pneumonia are alleged to be prone to the complication of hyaline muscle degeneration, a condition which has become known as Zenker's degeneration.

Rupture of the rectus abdominis muscle usually occurs in the weakest portion of the muscle—that below the semicircular line of Douglas, where the aponeurotic sheath on the posterior surface of the muscle is absent, and where the only posterior support is the transversalis fascia. Rupture of the muscle itself and of the inferior epigastric vessels would appear to be two indistinguishable conditions, as it is not easy to see how one could occur to any extent without the other.

The most interesting feature is the difficulty in diagnosis. Three of the four cases described below were admitted and explored as "acute abdomens." Diagnosis is particularly difficult in the fat patient, who, incidentally, is much more liable to the condition. The haematoma in the abdominal wall is so tender to palpation and movement that the only sign likely to be of value—tensing the abdominal muscles to fix the mass—is outside the patient's power of co-operation because of intense pain.

Most cases are sent into hospital labelled as twisted ovarian cysts or degenerating uterine fibroids. A number of cases have been reported in young people as a result of indirect violence, such as jumping over a gate or scaling a wall. The following four cases all occurred during coughing.

Case 1

A married woman aged 51 was admitted to hospital on January 23, 1946, with a history of sudden severe pain in the left iliac fossa, present since a coughing bout 36 hours previously. There were no other abnormal symptoms referable to any system, but just before admission she had noticed a lump in the left abdomen. There was also a previous history of three caesarean sections.

On examination her general condition was quite good, with a temperature of 97° F. (36.1° C.) and a pulse of 72. Abdominal palpation revealed a hard, fixed, fairly well circumscribed tender swelling in the left iliac fossa. A provisional diagnosis of twisted ovarian cyst was made and laparotomy carried out. A huge haematoma in the left rectus muscle was found, with disruption of the muscle. A fluted drainage tube was left in the cavity after shelling out the clot, and the abdomen was closed and the rectus sheath sutured. Short-wave diathermy was applied to the abdomen each day, and the patient was discharged 16 days after operation. When she was seen several months later the scar was sound and no clinical abnormality could be detected in the lower abdomen. It is possible that the scars in the lower abdomen predisposed to the rupture.

Case 2

A married woman aged 60 was admitted to hospital on October 21, 1946, complaining of sudden pain in the right iliac fossa several hours previously. This came on in a coughing attack; the cough itself had been present for a month.

On examination she looked rather ill and shocked. Her temperature was 97.8° F. (36.6° C.) and pulse 104. The abdomen was rather obese, but a large tender hard mass could be readily felt in the right iliac fossa. There appeared to be no generalized peritoneal irritation. A diagnosis of twisted ovarian cyst was made and laparotomy performed. On opening the right side of the rectus sheath a large haematoma was found, with disruption of the lower abdominal musculature. The clot was evacuated, no active bleeding-point was seen, and the muscle sheath was closed round a fluted drainage tube. Post-operatively, a course of sulphadimidine and short-wave diathermy was given, and the patient was discharged on the 20th post-operative day. The wound was rather slow to heal, but several months later the whole condition had completely resolved.

Case 3

A married woman aged 65 was admitted to hospital on October 18, 1947, with a history of severe pain in the left lower abdomen for four days, beginning when she had a severe coughing attack. She had vomited several times, and had not had her bowels moved since the pain began. There was nothing else relevant in the history.

On examination she looked ill, had a furred tongue, and there was a trace of jaundice in the conjunctivae. The pulse was 120, and the temperature 98.8° F. (37.1° C.). A hard tender swelling was present in the left iliac fossa, and this appeared to arise from the pelvis. A little bruising was noted at the umbilicus. On pelvic examination the mass could be pushed with some difficulty on to the examining finger.

The diagnosis of ruptured rectus was considered, but in view of the patient's poor general condition an intraperitoneal lesion, such as a perforated diverticulitis, could not be excluded, and therefore the abdomen was opened. There was a huge haematoma of the left rectus muscle, with free bleeding from the inferior epigastric artery, which necessitated its ligation just above the inguinal ligament. The clot was then shelled out, and the wound was closed round a drainage tube as before. A course of penicillin was given post-operatively, and the patient left hospital on the 12th day after operation with the wound well healed. Unfortunately, she did not report back for subsequent examination. A specimen of the muscle tissue from this case was reported as showing hyaline degeneration and infiltration of blood between the muscle fibres.

Case 4

A married woman aged 46 was admitted to hospital on November 11, 1948, complaining of sudden left lower abdominal pain following coughing. The cough had been present for one week, and she stated that her original sensation was of something snapping. There was nothing else of help in the history except that a hysterectomy had been performed abdominally one year previously.

On examination the general condition was good. Her temperature was 99.6° F. (37.6° C.) and pulse 72. A tender swelling was present in the left iliac fossa, and there were no signs of general peritonitis. Pelvic examination was negative. When the patient raised her head the mass became more fixed and tender, and this, coupled with the thinness of the abdominal wall, enabled a correct diagnosis of ruptured rectus abdominis to be made. Treatment was along conservative lines, with morphine and pethidine for the pain, and kaolin to the affected muscle. Later, short-wave therapy was given each day. When the patient was discharged eight days later the mass had disappeared, and on subsequent examination the abdomen was normal.

Summary

Four cases of rupture of the rectus abdominis muscle are reported, all occurring in non-pregnant women during an attack of coughing. Three were treated by exploration and one conservatively. The recovery in each case was good, irrespective of the method of treatment. The difficulties of differentiating the condition from twisted ovarian cyst or strangulated interstitial hernia are mentioned, and the value of short-wave therapy in the post-operative phase is emphasized. Finally, it would appear that lower abdominal scars predispose to the condition.

My thanks are due to Mr. F. W. Duthie, Mr. L. M. Zinck, and Mr. J. Grocott for permission to publish these cases.

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Spontaneous Haematoma of the Rectus Abdominis Muscle in Pregnancy

In 1882 Karl Maydl, of Vienna, wrote a most valuable series of articles on the subcutaneous tearing of muscles and tendons. He devoted much attention to the tearing of the rectus abdominis muscle and tabulated his cases. In 1918 Emerson recorded a case of rupture of the deep epigastric artery due to muscle strain. Perman was the first to write a comprehensive article on haematoma in the sheath of the rectus abdominis muscle. In 1925 Carey Culbertson reported two cases of "haematoma occurring spontaneously in the sheath of the rectus abdominis muscle, with consideration of its gynaecological and obstetrical significance." In 1937 Cullen and Brödel, of the Johns Hopkins Hospital, reported two cases of spontaneous haematoma of the rectus abdominis during pregnancy. Both were coloured patients. Resolution was uneventful in one with a seven-months pregnancy, but operative exploration was carried out on the other with success. Torpin in 1943 analysed 27 reported cases. In 1946 Teske reported a case and analysed 100 cases from the literature. Aird (1949) stated that only about 150 cases have been reported to date.

Case Report

A 4-para aged 38 was admitted to hospital on January 30, 1950, complaining of pain and swelling in the peri-umbilical region on the left side. She was in the 29th week of pregnancy. She had had a cough for 10 days, worsening in the last two days before admission. The pain and the mass appeared immediately after a bout of coughing just half an hour before admission.

On examination a large swelling was easily visible on the abdominal wall. It was 3¾ in. (9.5 cm.) long and 2¼ in. (5.7 cm.) broad, with its long axis vertical over the medial half of the left rectus and immediately to the left of the umbilicus. The medial edge of the fluctuant mass was 1 in. (2.5 cm.) away from the umbilicus. There was moderate rigidity and tenderness along the whole length of the left rectus. The foetal heart and pregnancy were quite normal.

A clinical diagnosis of spontaneous haematoma of the left rectus was made, and rest in bed with recumbency was ordered. The haematoma was not interfered with, but close observation of the progress of the resolution of the mass was made.

Shock was slight on admission and passed off within one and a half hours. Rigidity and tenderness of the rectus disappeared within four hours. Ecchymosis and longitudinal staining of the skin appeared along the length of the left rectus for about 6 in. (15 cm.). The spindle-shaped area of stain was about 2 in. (5 cm.) wide at its centre but tapered towards each end. Blue staining of a crescentic area at and immediately below the umbilicus appeared 24 hours after admission. It measured ¾ in. (1.9 cm.) long and about ¼ in. (0.6 cm.) wide. It changed to a dark brown discoloration after two days, when a diffuse light brown area appeared along the medial edge of the left rectus. The swelling and staining had completely disappeared by the 13th day. The pregnancy proceeded normally to term and she was delivered on April 26 of a normal 7¼-lb. (3.3-kg.) child.

Discussion

This condition occurs mainly in multipara and late in pregnancy. It has also been described in the puerperium and even within half an hour of delivery and expulsion of the placenta. In pregnancy it may be that the overstretching of the muscle increases its susceptibility to trauma.

In almost all cases in pregnancy there was some evidence of trauma from labour, from coughing, or from a fall. The trauma of coughing was important in the cases of Abelin, Andrews, Ashkar, Camelli, Carreti, Casu, Cullen, Decio, Edwards, Hobbs, Schroeder, Stoeckel, Torpin, and in the case here reported. Muscle degeneration from influenza or typhoid is a possible cause.

The precipitating factor in most spontaneous cases seems to be inelasticity of the wall of an artery or vein which prevents the vessel accommodating itself in a movement, a cough, or a sneeze; or, in or after labour, to the remarkable variations in length which the rectus muscle undergoes between extreme contraction and extreme relaxation. According to Brödel there is only one major vessel to take care of this long stretch of muscle, and in order to avoid damage to itself it must keep away from the muscle so far as is possible and send its branches into the muscle substance in such a manner that the muscle action does not interfere with vascular freedom. The larger intramuscular arteries branch freely and form numerous anastomoses. They run at an angle varying from 60 to 90 degrees to the axes of the muscle bundles. If an occasional artery runs parallel to the muscle bundle it shows greater tortuosity.

The arteries are less apt to tear than the veins, because they are far more resistant. They lie so loosely embedded in the intramuscular connective tissue that they can be pulled out quite far without injury; but not so with the veins. They are frail, of smaller calibre, and have a much thinner wall. Tears may be partial or complete. In a partial tear only branches of the main vessels rupture beyond their point of entrance into the muscle body, but a complete tear is apt to rupture the main trunk also.

Among the clinical findings premonitory vague discomfort at the site of bleeding is common with the lesser degrees of haemorrhage. Probably a great number of the haematomata are small, unnoticed, and consequently unreported. In most recorded cases the haematoma was limited to the rectus sheath, and pain and tenderness were felt over the bleeding area. Muscle rigidity is often marked in the involved rectus, but in small haemorrhages there is little more than a local tenseness in the muscle. Swelling is usually confined to the affected muscle sheath. In some cases the haemorrhage escaped the limits of the muscle sheath and extended into the broad ligament or ruptured into the peritoneal cavity. In two cases in the literature death occurred before help arrived. The haemorrhage lies initially between the fascia transversalis and the posterior surface of the rectus muscle, spreading later to surround the muscle and ascend over the linea semilunaris between the rectus muscle and its posterior sheath and sometimes downwards behind the rectus. If the patient lifts her head off the pillow so as to contract the rectus the mass can be felt confined to the rectus sheath and immovable.

Staining of the skin and ecchymosis are very common. They occur over the centre of the haematoma, as a semicircle around the umbilicus, above the pubis, or along the linea alba. The extravasated blood can pass more easily superficially around the medial border of the rectus. The pigments that produce this phenomenon may reach their destination by following the ordinary fascial planes but not by the lymphatics as was previously believed.

Where the haematoma was small it usually resolved uneventfully within a week or two. Where the haemorrhage was extensive the diagnosis was often confused with intra-abdominal lesions, a laparotomy was done, and the condition was found when the rectus sheath was opened. Repair of the lesion was followed by recovery in some cases, but rupture of the wound and peritonitis and death of the foetus occurred in others. Some cases not operated on showed persistence of the cystic area of haemorrhage, and calcification followed in one. In a few cases the haematoma ruptured into the peritoneal cavity.

This lesion has carried a very high maternal and foetal mortality even since publication of clinical details of the first cases. Hobbs's patient died in less than an hour after the haemorrhage began and before blood transfusion could be started; necropsy revealed 3 pints (1.7 litres) of blood in the sheath of the left rectus, with one large recent tear in the posterior surface of the muscle at the junction of the middle and lower thirds and evidence of older tears and haemorrhages in the upper segments. In Torpin's series there was a 15% maternal mortality and a 50% foetal mortality. Recent reports show a maternal mortality of 12% and a foetal mortality of 25%. Most of this loss of life could have been obviated by correct diagnosis.

Many patients have been referred for surgical opinion with a presumptive diagnosis of acute appendicitis. In others an acute obstetrical emergency had been presumed. Operative intervention is often unnecessarily urged, with the grave extra risk of damage to the foetus. Relief from the intermittent strain of coughing, with rest in the recumbent position and adequate treatment for shock, are the essential points in treatment. If the haematoma is very extensive and when shock is controlled, a paramedian incision with section of the rectus sheath and retraction of the muscle laterally will facilitate visualization of the bleeding vessel. Suture of ruptured vessel or muscle is simple. Drains should not be used.

I wish to thank Dr. Gerald Connolly for his permission to record this case from the maternity department of the hospital.

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In addition to treating "real" accident victims at the South Bank the Red Cross rendered first aid to many others (*The Times*, October 19). Sixteen of the 11,000-odd patients treated by the Society during the exhibition are classified in the log-book under the heading of "immersion," and when rescued from the ornamental lakes they found refuge with the Red Cross. First aid was given to a cow with toothache, and she was able to return to her stall in the agriculture exhibition. The first-aid unit was staffed by 308 men and 321 women from all over the country, many of them giving up their holidays for this purpose.

Reviews

PROBLEMS OF SEX

The Child Unborn. By R. J. Harrison, M.A., D.Sc., M.B., B.Chir., F.L.S. (Pp. 217; 60 illustrations. 15s.) London: Routledge and Kegan Paul. 1951.

Any Wife or Any Husband. A Book for Couples who have met Sexual Difficulties; and for Doctors. By Medica. (Pp. 159. 7s. 6d.) London: William Heinemann. 1950.

Contraceptive Technique. A Handbook for Medical Practitioners and Senior Students. By Helena Wright, M.B., B.S.(Lond.), with the assistance of H. Beric Wright, M.B., B.S.(Lond.). (Pp. 68; 16 illustrations. 6s.) London: J. and A. Churchill, Limited. 1951.

Woman: Her Change of Life. By Miriam Lincoln, M.D., F.A.C.P. (Pp. 116. 6s.) London: Williams and Norgate, Limited. 1951.

The causes of marital breakdown and disharmony are many, but there is little doubt that sexual difficulties often play a direct or indirect part. In the hope of avoiding these and allied problems, a recent trend has been to encourage education in the physiology of reproduction from a comparatively early age. Elementary instruction in these matters now forms a part of the curriculum of many schools, and other official bodies, such as the Marriage Guidance Council, are concerned to extend this education to older people before and after marriage. Many people, however, prefer to get the information by reading rather than from personal interview with a doctor or counsellor or friend; for these, several books on various aspects of sex are available. Dr. R. J. Harrison contributes another in *The Child Unborn*, but this is different from most in the amount of detail it gives. It is an accurate and complete account of the physiology of reproduction, and contains many interesting references to comparative anatomy and physiology. The author introduces it with a plea for an extension of knowledge on these matters to the community at large, but unfortunately he has written it at such a level that only a few of the community—those with a scientific training and an ability to understand the numerous photomicrographs—will be able to appreciate it. The rest are likely to find the wealth of technical terms and the mass of detail too much for them. Moreover, no glossary is included to help. It is a pity that this book, which is excellent in so many ways, is likely to fall between the layman who expects less, and the medical student and doctor who expect more.

This raises the question of how much should be explained to the uninitiated. If a couple on the eve of marriage are told of all the various sexual difficulties they may encounter, it may create unnecessary anxiety and produce the very conditions which lead to these difficulties. Love-making is, in the words of the popular song, "doin' wot comes naturally," and, left to themselves, only a small number of couples will find that it does not come naturally. So in regard to *Any Wife or Any Husband* the emphasis should be placed on the subtitle, "A Book for Couples who have *Met* Sexual Difficulties; and for Doctors." The author, who remains anonymous, is both a doctor and a grandmother, and this varied experience stands her in good stead. She writes simply and clearly of the psychological and physical sexual difficulties which may confront either man or woman. She includes impotence, frigidity, and their opposites, and gives a common-sense picture, with short accounts of illustrative cases. Although diagnosis receives more attention than treatment, any doctor should find this small and cheap book of the greatest value in handling the intimate problems of his patients, and should have no hesitation in lending it to the more intelligent of them.

Contraception looms large in the picture of domestic bliss, especially in these days of economic stringency and shortage of help in the home. How many marriages have become humdrum and embittered because children have arrived too often and in too large a number? How often is dyspareunia traceable to fear of pregnancy? How often is nervous tension due to sexual abstinence or coitus interruptus? Instruction in a reliable method of contraception may make all the difference to the physical and mental well-being of a married couple, and any doctor who wishes to give this service to his patients will find the two or three best methods well and simply stated in *Contraceptive Technique*, by Dr. Helena Wright. This book again is suitable for lay readers, and the busy practitioner may be glad to take advantage of this.

Marriages which survive the problems of the reproductive age may still founder on the rocks of the menopause. Once past the age of 40 years many women live in dread of the change of life, and, in consequence of this fear more than from the effect of the climacteric itself, allow themselves to become so upset that they disrupt the household for many years. When menopausal symptoms are severe, however, they are most distressing, and it is important that the husband as well as the wife appreciates the position and exercises tolerance. In these matters Dr. Miriam Lincoln takes the view that to be forewarned is to be forearmed; that if women and their husbands know what to expect, and that if their mostly ill-founded fears are put at rest, the change of life will come about all the more smoothly. She emphasizes that in 75% of cases the menopause does not cause enough disturbance to warrant even taking medical advice, and that hormone therapy is necessary in only 8% of cases. To put this book to the test I asked an intelligent woman, aged 42, who was convalescing from an operation involving removal of both ovaries, to read it. She wrote the following comment: "Not having read any similar books, I cannot say it is the best of its kind, but it strikes me as being sensibly and clearly written, and it would be a help to any woman requiring reassurance. I should imagine it might most profitably be read by general practitioners—particularly male—as, being written by a woman, it gives a good insight into the average female mind in relation to these matters." She added verbally, "And gynaecologists as well as general practitioners." I agree.

One of the difficulties about giving instruction on matters concerning sex and marriage is that it is time-consuming, every case requiring individual attention. For this reason as much as any other the general practitioner and gynaecologist have not always given their patients all the help they might. As a result, and in addition to marriage guidance centres, which deal with legal and more general aspects of the problem, there have sprung up in recent years birth control clinics and sterility clinics and combinations of the two.

So far as contraception is concerned this arrangement is convenient, and, although not ideal, will continue to fill a real gap until this subject is more widely taught in medical schools. In regard to infertility and other sex problems, however, the situation is different, and

facilities for dealing with these are already available. Moreover, special infertility clinics, other than those forming part of a regular gynaecological out-patient service, have serious disadvantages, for they can easily become pseudoscientific, and they in any case tend to give the problem disproportionate emphasis in the minds of the patients, doctors, social workers, and administrators. They may, in fact, cause rather than cure marital disharmony and individual unhappiness.

"Medica" goes so far as to advocate the setting up of family welfare centres under the auspices of the National Health Service, such centres to offer advice on sexual disorders, sterility, and contraception, while Dr. Helena Wright puts in a strong plea for support of the Family Planning Association, which now includes infertility and pregnancy diagnosis within its ambit. Most of those with experience in these matters will regard any such developments with alarm, and may envisage that it may not be long before there is a demand for the creation of consultants in birth control or in infertility, and for the appointment of sexologists.

There is everything to be said for concentration on infertility by a few research workers, but little to be said for the wholesale establishment of clinics for dealing with problems which are essentially ones for personal discussion between the family doctor and the family, the doctor being able to call freely on hospital and consultant services which are already equipped to give balanced scientific advice and treatment in cases which need it. It may well be that medical students require more instruction in some of these matters, and that in certain parts of the country the present hospital facilities need some elaboration. To remedy these deficiencies would be comparatively easy and would probably offer more to family welfare than would the setting up of additional special clinics.

T. N. A. JEFFCOATE.

THE ENTEROBACTERIACEAE

Enterobacteriaceae. Collected Studies on Salmonella, Arizona, Ballerup-Bethesda, Escherichia, Alkalescens-Dispar, Klebsiella, Shigella, Providence, and Proteus. By F. Kauffmann, M.D. (Pp. 338. Sch. 50.) Copenhagen: Ejnar Munksgaard. 1951.

In recent years much attention has been paid to the family Enterobacteriaceae, the classification of which is based on the biochemical reactions and serological relationships of the various groups within the family. The author is recognized as an authority of international repute on this subject, to which he has contributed much, and this book, as stated in the subtitle, is the result of his personal studies and of work done in his laboratory. Most of this work has been published by him in scientific journals, and some is confirmation of the published work of other authors. It is a pity in such a comprehensive survey of the modern work on this subject that comparatively little reference has been made to older classifications and work, but no doubt this is explained by the subtitle.

There are various criticisms which can be mentioned, such as the use of the symbol V both for the Vi antigen of the typhoid bacillus and for the serologically unrelated V (Roman 5) somatic antigen found in many *Salmonella* species. The author gives his method for the testing of patients' serum for the presence of typhoid Vi agglutinin; the suspension and method which he recommends differ from those accepted by the expert committee on standardization of the World Health Organization. The author also states that in the serological identification of some groups of organisms slide-agglutination technique, with certain provisos, makes tube agglutination superfluous. Though this is true of specialist laboratories, it is not true of the routine laboratory which investigates comparatively few strains, usually at relatively long intervals; these laboratories should confirm their results by tube agglutination.

In many instances the same antigen is found in different biochemical groups. It is confusing to find that such an antigen is referred to by numbers which differ according to the biochemical classification. Though there are certain practical advantages in this method, it is confusing to the newcomer even though a table of equivalents is given on p. 296 of the book. The author states that many organisms show some characteristics of more than one group, and he has made no attempt, perhaps wisely, to fit these into his classification. Nor does he mention the source of the strains, and he gives little useful information on their relationship to disease processes.

In spite of these criticisms the book is a most valuable collection of information which has hitherto appeared only in scattered papers. It will be of most use to those engaged in research on these and other organisms rather than to those working in routine laboratories.

JOAN TAYLOR.

FIRST PRINCIPLES IN PSYCHOLOGY

Handbook of Psychology. By John H. Ewen, F.R.C.P.Ed., D.P.M. (Pp. 216. £1 5s.) London: Sylviro Publications. 1951.

When a medical man wishes to learn something about psychology for use in the clinic or in self-defence at the dinner table, it is not easy to advise him how to start. If he begins with the history and basic concepts of the subject, he may have mental indigestion before he comes upon anything which can be applied to his daily life and work. If, on the other hand, he starts with abnormal psychology, he may be uncritical of current theory through lack of knowledge of the general background. Probably the best method is to begin with a general guide which will give him the first principles and their relation to the many fields of applied psychology. To provide this is Dr. Ewen's aim, and he succeeds in covering a great deal of ground.

Although the book is not a long one, it reads slowly because of condensation and because the subject-matter calls for close attention. The greater use of italics and subheadings might have helped to break up the text. There is rather too much for the needs of the average medical student, although he will benefit greatly from reading it at a time when pressure of examinations is not hampering his capacity for absorbing principles as opposed to facts. For the postgraduate the book provides a useful digest, and a short bibliography is given for further reading. Its use as an introduction to the subject would, however, be more effective if references were given to the innumerable authors whose names are quoted.

The ground covered is mainly the traditional basic literature of psychology, with reference to recent work and problems of current interest. In the final chapters the author surveys the application of psychology to clinical problems so that the reader is introduced to the language and methods of psychological medicine. The price seems high for a handbook of this size.

ALEXANDER KENNEDY.

FIFTY YEARS OF THE R.C.S.

Royal College of Surgeons of England. A Record of the Years from 1901 to 1950. (Pp. 80. Paper covers, 5s.; bound in maroon book-cloth with gold lettering, 8s. 6d.) London: Royal College of Surgeons of England. 1951.

This little book of eighty pages gives a brief history of the Royal College of Surgeons for the first fifty years of this century, followed by short lives of the sixteen presidents "who have directed its policy during those years." The record cannot fail to be of interest to all associated with the College, and certainly every Fellow should possess a copy.

In 1900 the College was a dignified and respected body which conducted examinations and possessed a magnificent museum and an excellent though usually almost deserted library; there was a minimal amount of research done, and only a small amount of teaching beyond the annual eponymic lectures. Now the College has a constantly used and thronged library, splendid research departments, crowded classrooms where renowned professors instruct eager students drawn from all parts of the Commonwealth, and a beautifully equipped hostel where some of the students live. This booklet tells you how this change came about and who were those responsible for the changes.

Each of the lives of the presidents is a thought-provoking success story, and the reader may find some interest in estimating what factors most contributed to the attainment of this exalted position. The only common factors appear to be great natural ability and the will to succeed. It will be noted, however, that those most responsible for the recent increase in usefulness and prestige of the College possessed remarkable administrative ability and fertility of resource. We wish it had been possible to precede the life of every president by a photograph of the subject of the memoir.

Those who are fond of facts will be interested to know that the average age at which the presidency has been attained is 62.4 years. Though some presidents were regarded as delicate, the average length of life was over 75 years. We do not think that every Fellow could say which president was born at sea and later became consulting surgeon to the Navy; and we are sure it would surprise many to learn how seldom has a graduate of Oxford or Cambridge University attained the presidency of the Royal College of Surgeons.

V. ZACHARY COPE.

Since its discovery in 1922 vitamin E has been the object of considerable research on the part of the physician and the chemist. In 1941 Merck and Co. published an annotated bibliography of work on the subject prior to 1940, and Distillation Products Industries (a division of the Eastman Kodak Company) have now made available a similar bibliography to cover the years 1940–50, during which period there were published more than 1,500 scientific papers and patents concerned with vitamin E. This *Annotated Bibliography of Vitamin E, 1940 to 1950* (Distillation Products Industries, Rochester, 3, N.Y., U.S.A. 184 pp. $3. 1950) is classified by subjects, and the brief abstracts accompanying most of the references emphasize the physiological effects of vitamin E rather than its chemical aspects and clinical application. An author index is provided. The book is reproduced by a photo-offset process.

BOOKS RECEIVED

Review is not precluded by notice here of books recently received

Venereal Diseases. By R. C. L. Batchelor, M.A., M.B., Ch.B., D.P.H., F.R.C.S.Ed., M.R.C.P.Ed., and M. Murrell, M.B., B.S., D.P.H., F.R.C.S.Ed., M.R.C.S. (Pp. 217. 12s. 6d.) Edinburgh: E. and S. Livingstone. 1951.

Applied Anatomy for Nurses. By E. J. Bocock, S.R.N., S.C.M., D.N., and R. W. Haines, M.B., D.Sc., F.L.S. (Pp. 320. 15s.) Edinburgh: E. and S. Livingstone. 1951.

The Essentials of Modern Surgery. Edited by R. M. Handfield-Jones, *M.C.*, M.S., F.R.C.S., and Sir Arthur E. Porritt, K.C.M.G., C.B.E., M.A., M.Ch., F.R.C.S. 4th ed. (Pp. 1,263. 55s.) Edinburgh: E. and S. Livingstone. 1951.

A. F. Holleman's Organic Chemistry. Revised by J. P. Wibaut. (Pp. 660. 55s.) London: Elsevier Publishing Company. 1951.

And They Shall Walk. The life story of Sister Elizabeth Kenny. (Pp. 267. 15s.) London: Robert Hale. 1951.

Ambulation. By K. A. Dening, B.S., M.Ed., F. S. Deyoe, Jun., B.S., and A. B. Ellison, B.S. (Pp. 188. 27s. 6d.) London: Mayflower Publishing Company. 1951.

Diseases of Infancy and Childhood. By W. Sheldon, M.D., F.R.C.P. 6th ed. (Pp. 812. 40s.) London: J. and A. Churchill. 1951.

Clinical Tropical Medicine. Edited by R. B. H. Gradwohl, M.D., and others. (Pp. 1,647. 160s.) London: Henry Kimpton. 1951.

The Common Sense of Science. By Dr. J. Bronowski. (Pp. 154. 8s. 6d.) London: William Heinemann. 1951.

Your Hayfever and What To Do About It. By H. Swartz, M.D., F.A.C.A. (Pp. 182. 21s. 6d.) New York: Funk and Wagnalls Co.

The Electrical Activity of the Nervous System. By M. A. B. Brazier, B.Sc., Ph.D. (Pp. 220. 25s.) London: Sir Isaac Pitman. 1951.

The Mode of Action of Anaesthetics. By T. A. B. Harris, M.B., B.S., D.A., F.F.A.R.C.S. (Pp. 768. 42s.) Edinburgh: E. and S. Livingstone. 1951.

Pharmacology and Therapeutics. By A. Grollman, Ph.D., M.D., F.A.C.P. (Pp. 828. 70s.) London: J. and A. Churchill. 1951.

IV^e Congrès Neurologique International. Vol. 3. (Pp. 505. No price.) Paris: Masson. 1951.

Les Principales Positions Utilisées en Radiographie. By L. Delherm and H. Morel Kahn. 3rd revised ed. (Pp. 290. 1,800 francs.) Paris: Librairie Maloine. 1951.

11^e et 12^e Sessions de Conférences du Comité International de Médecine et de Pharmacie Militaires. (Pp. 120. No price.) Liége: Office International de Documentation de Médecine Militaire. 1951.

Les Xanthomatoses. By A.-L. Froehlich. (Pp. 311. No price.) Brussels: Acta Medica Belgica. 1951.

Estados Endocrinopáticos Agudos. By Dr. M. T. Torm. (Pp. 134. No price.) Barcelona: Byp. 1951.

Hirnatrophische Prozesse im Mittleren Lebensalter. By Dr. F. W. Bronisch. (Pp. 105. M. 12.60.) Stuttgart: Georg Thieme. 1951.

Meister der Chirurgie und die Chirurgenschulen im Deutschen Raum. By Professor H. Killian and G. Krämer. (Pp. 232. M. 24.80.) Stuttgart: Georg Thieme. 1951.

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PSYCHOSOMATIC MEDICINE

With the ever-increasing burden of psychosomatic literature, mainly originating in America, it is important to pause and review the contribution being made to medicine by this movement. It is essentially a reaction against thinking of disease only in such terms as bacterial invasion, hormonal imbalance, or physical change in the body. Psychosomatic medicine implies that disease may be the sequel of nervous or emotional stress—for example, a maladaptation, or loss of power of bodily compensation, to increased autonomic stimulation from the higher centres. The effects of nervous stress would depend on the emotional load, the personality of the individual, and the general level of mental and physical health. Gastric and duodenal ulcer, essential hypertension, coronary artery disease, hyperthyroidism, migraine, colitis, and many others are listed by the psychosomatic authorities as within their sphere.

How much of this rests on a solid foundation and how much is due to the missionary zeal of those converted to a new faith? That emotions can affect the heart, stomach, bowels, and skin is common experience. The striking effects which may be seen on secretion, vascularity, and motility of the viscera are becoming well known, and first-class scientific studies have been recorded, particularly by Wolf and Wolff.[1] The second link in the argument is that such physiological disturbance may lead to organic disease, either by aggravating an existing process or by initiating a new one. Clinical experience provides clear evidence of the dramatic association of acute emotional disturbance with the onset of symptoms or relapse in some patients suffering from one of the psychosomatic group of diseases. Striking case histories are recorded in profusion in the medical literature—e.g., acute anxiety preceding haemorrhage from, or perforation of, a peptic ulcer,[2] or of great grief preceding the onset of ulcerative colitis.[3] Even the rapid development of obesity has been described following emotional trauma.[4]

It is, however, the exception rather than the rule to be able to relate emotional catastrophe to the onset of one of these diseases. This, at least, is the experience of most practising physicians even if they are particularly interested in the subject. Some papers claim a high proportion of associated environmental upsets, but such high figures tend to include the lesser difficulties of day-to-day life. Clearly another factor is needed to explain why some people break down from relatively trivial difficulties while others may withstand the worst totalitarian injustice and yet remain free from psychosomatic disease. The difference may lie in the personality of the psychosomatic subject compared with the general population. It is easy to appreciate that with some personalities emotional stress may become magnified—e.g., the dependent, sensitive individual will, when circumstances shatter his world, suffer far greater nervous strain than the independent phlegmatic man. It is at first sight an attractive hypothesis, and much has been written on the constellation of personality facets in psychosomatic disease, but in spite of the volume of literature on this point there is no real evidence that groups of patients with hypertension or peptic ulcer differ in any way from the general population.

The many studies of personality made without controls are meaningless, but where controlled investigations have been made they lend no support to the existence of personality patterns. For example, a careful study of hypertension[5] showed no significant difference in the assessments for personality facets between "neurogenic" hypertension and for other types (endocrine or renal). Brown,[6] using a Rorschach test, compared 25 patients with ulcer with 25 controls. The observer was unaware of the identity of the patient and found no difference in the mean score, although the ulcer group was more homogeneous. Hamilton,[7] using a questionary devised to elicit personality traits said to be common in sufferers from peptic ulcer, failed to show any difference between patients with peptic ulcer and the controls. In view of the stress placed by many writers on the role of childhood in determining a personality liable to the group of diseases mentioned, it is surprising that so little factual information exists on this point. In a paper appearing elsewhere in this issue Dr. T. D. Kellock reports that he found no significant difference in the childhood experience of 250 patients with duodenal ulcer compared with 250 controls. Admittedly his observations related to the frequency of grave misfortune in childhood, such as early death of a parent, and a more sensitive index of the inter-family

[1] *Gastroenterology*, 1950, **14**, 93.
[2] Davies, D. T., and Wilson, A. T. M., *Lancet*, 1939, **2**, 723.
[3] Groen, J., *Psychosom. Med.*, 1947, **9**, 151.
[4] Shorvon, H. J., and Richardson, J. S., *British Medical Journal*, 1949, **2**, 951.
[5] Gressel, G. C., Shobe, F. O., Saslow, G., Dubois, P. H., and Schroeder, H. A., *J. Amer. med. Ass.*, 1949, **140**, 265.
[6] *Psychosom. Med.*, 1950, **12**, 1.
[7] *Brit. J. med. Psychol.*, 1950, **23**, 182.
[8] Paulley, J. W., *Gastroenterology*, 1950, **16**, 566.
[9] Groen, J., *Psychosom. Med.*, 1947, **9**, 151.
[10] White, B. J., *New. Engl. J. Med.*, 1951, **244**, 537.

relationship may be needed. There is still a possible case for patients with colitis having specific personality traits as described by various writers,[8 9] but it is at least as likely that the unpleasant nature of their illness may modify their personality pattern, and indeed a striking change during remission has recently been recorded.[10] It may be argued that only a psycho-analytical approach can reveal fundamental differences of personality, but any satisfying studies of psychosomatic disease remain remarkably difficult to discover in the medical literature.

That emotional factors may cause disturbance in bodily function and that the intensity of emotional trauma may be modified by personality is indisputable, but no convincing evidence exists to prove that psychosomatic groups of diseases may be acquired by virtue of a specific personality. Nor is there any convincing evidence that emotional stress initiates a disease process, although it undoubtedly aggravates an existing one. There is a possibility that in a small number of individuals intense emotional disturbance might lead to physical change—e.g., an acute gastric ulcer causing haemorrhage—but it is equally likely that an acute ulcer already present had been stimulated into further activity. Instead of regarding emotional tension as a major aetiological factor in its own right, it may best be regarded as an aggravating factor in the same category as, for example, fatigue or over-smoking. It is well known that nervous tension may exert a profound effect on the endocrine system, and indeed it would be remarkable if directly or indirectly it should not modify an existing disease process.

Even if psychosomatic factors merely aggravate, rather than initiate, organic disease, nevertheless the psychosomatic school of medicine has made a useful contribution to contemporary thought. Psychosomatic medicine essentially is not a specialty but a point of view, and an approach which applies equally to medical and surgical problems. It is basic medicine, which has needed re-emphasis. The compassionate aspect of medicine has perhaps been rather overshadowed by the rapid growth of techniques of investigation and by the present abundance of specific remedies. The best physicians and surgeons have long regarded their patients as individuals with backgrounds which may influence their illnesses. Some medical men, because they have been too hurried or too poorly trained, may have tended to think only in terms of the immediate disease process and have not had a sympathetic understanding of the patient's problems. There is no doubt that many patients benefit merely from the taking of a detailed history at leisure and in privacy, and the unburdening of their story, often untold to others, may save many weeks of attendances at out-patient departments or surgeries. But "psychosomatic" is an unnecessary term, and the sooner it is dropped from the current literature the better.

PROTOZOA VERSUS MANKIND

While the purely medical importance of the protozoal diseases of man is well known, the extent of the economic havoc they cause is perhaps not so widely realized. The effect of protozoa on human affairs is seen in various spheres of activity, but it is only among their free-living representatives that forms occur which are beneficial, since they contribute something that can be utilized by man, whereas most of the parasitic protozoa are harmful. Free-living protozoa can be of help to, among others, geologists, mining engineers, public health workers, and fishermen. Thus, fossil Foraminifera enter into the composition of limestone, which provides building materials, while in the petroleum industry these rhizopods furnish valuable clues to oil-bearing zones. Various coprophilic protozoa are of public health importance, because of the part they play in promoting sewage purification and as indicators of pollution in water supplies. Finally, planktonic marine protozoa are a source of food for fish. However, the harmful effect of parasitic protozoa upon the economic life of man far outweighs the usefulness of the free-living forms. Apart from human protozoal infections, some of the protozoal diseases affecting domesticated animals and useful wild animals—such as food, fur-bearing, and game animals—are of practical importance in animal husbandry, fisheries, agriculture, sericulture, apiculture, and other branches of economic zoology.

One of the vital problems of our age is to grow enough food for an increasing world population. This necessitates not only a progressive expansion of agriculture and animal husbandry but also the protection of crops and food animals from losses due to diseases, among which those caused by protozoa are often devastating. The bearing of protozoal diseases upon human prosperity is discussed in this issue by Professor H. E. Shortt, who has selected two diseases affecting cattle—trypanosomiasis and piroplasmosis—and human malaria to illustrate the impact of protozoa on world economic problems. In West Africa alone bovine trypanosomiasis is believed to cause an annual loss of more than £1,000,000 through the death of livestock. This diminishes the vitality of the native population, because of the serious deficiency of their diet in high-grade protein and a steady deterioration of the fertility of the soil in the absence of beasts. These factors are causing a progressive

impoverishment of the people, while medical and administrative policies are encouraging a steady growth of the population. There are also incalculable indirect losses from the exclusion of countless numbers of livestock from potential pastures which at present are infested with tsetse flies and game animals harbouring trypanosomes. The restrictions thus imposed lead to overstocking of available grazing-grounds, with consequent soil erosion. From Professor Shortt's account it is evident that in Kenya alone East Coast fever is responsible for more than 50% of all parasitic infections in a cattle population numbering between five and six million. The mortality in cattle is more than that from all other causes put together, reaching 95% in South Africa.

These examples of the colossal economic damage caused by protozoal infections of domestic animals relate only to one host in selected localities. To these could be added many other examples. In the United States, for instance, the annual losses due to protozoal infections have been estimated at $10,000,000, half of which is attributed to poultry coccidiosis, a disease which is prevalent throughout the world, including this country. Another protozoal disease which may assume epidemic proportions is bovine anaplasmosis, which causes annual losses in the United States amounting to $100,000.

More directly disastrous to mankind are the human protozoal diseases. In India there are thought to be about one hundred million cases of malaria every year, with one million deaths. Wherever this disease is prevalent the health of the people may be undermined to such an extent that the welfare of the whole community is endangered, with calamitous effects on agriculture, industry, and the social services. The urgency of appropriate counter-measures of control and treatment becomes ever more pressing, but, as Professor Shortt points out, interference in the complex biotic relations between man and his environment may have serious and far-reaching consequences. Thus agricultural projects such as rice cultivation have brought about a temporary increase of malaria in parts of West Africa, and it is feared that one consequence of introducing any wet crops in Africa will be increased breeding of *A. gambiae* unless there is close supervision.[1] Again, the reclamation of a locality by eradication of trypanosomiasis or piroplasmosis might lead to its over-stocking and irreversible soil erosion. Piecemeal planning may do more harm than good, and measures of control must be carefully thought out by teams of doctors, veterinary surgeons, and agricultural experts, working in collaboration with biologists versed in animal and plant ecology.

[1] *World Hlth Org. Tech. Rep. Ser.*, No. 39, 1951, Geneva.

HOW DRUGS ACT ON TRYPANOSOMES

The science of chemotherapy was founded in the first place on the study of drugs which would kill trypanosomes, the protozoal parasites causing African sleeping-sickness. Their study is still of great importance, partly because trypanosomes are closely similar to the spirochaete of syphilis, which is less easy to handle in the laboratory, and partly because they are convenient for fundamental studies which may yield conclusions applicable to many other branches of chemotherapy. The discovery of an effective chemotherapeutic remedy is a problem of selective toxicity. Many compounds are known which are highly toxic to trypanosomes, but most of these must be rejected because they are also toxic to the host. The accepted drugs are compounds which have been chosen because they poison the parasite without poisoning the patient. The possibility of such selective toxic action depends on the existence of differences between the chemical reactions of trypanosomes and those of man.

The various trypanocidal compounds can be classified into certain broad groups according to their type of action. One such group is formed by the well-known arsenical compounds (such as neoarsphenamine), acriflavine, and stilbamidine. These compounds are highly active against the trypanosomes *in vitro*, and when given to infected animals they kill most of the parasites in a few hours. They are avidly absorbed by the trypanosomes, so that the concentration inside the parasite becomes several hundred times as great as that outside, and thus the compound becomes well placed to exert its toxic action.[1] The arsenicals have a selective combination with sulphydryl groups, which probably blocks important systems of enzymes involving glutathione and similar compounds.[2]

Another group of compounds is formed by suramin, phenanthridinium compounds (for example, dimidium bromide), and "antrycide," which resemble each other in many aspects of their biological activity. But they do not all act on the same point in the parasite, since a trypanosome which has been made resistant to one of them is not always resistant to the others. These compounds have no significant trypanocidal action *in vitro*, and when administered to infected animals they take one or more days before they remove the parasites from the blood. They are absorbed by the trypanosomes in such small amounts that absorption is difficult to demonstrate by ordinary chemical methods. These otherwise inactive compounds, however, possess the remarkable property of making the trypanosomes non-infective to other animals. After the trypanosomes have been in contact with the drugs *in vivo* or *in vitro* for a few hours they are apparently unchanged, and they continue to live and wriggle actively for at least a day. But if they are injected into a fresh mouse the usual acute infection fails to appear. It is almost as if the trypanosomes have lost their power of multiplication, so that when individual parasites are gradually destroyed

[1] Hawking, F., *Ann. trop. Med. Parasit.*, 1939, **32**, 313.
[2] Peters, R. A., Sinclair, H. M., and Thompson, R. H. S., *Biochem. J.*, 1946, **40**, 516.
[3] *Brit. J. Pharmacol.*, 1951, **6**, 325, 334.

by the defence mechanisms of the body they cannot be replaced by new ones (though the explanation is probably not quite so simple as this).

Fresh light has been thrown on this phenomenon by some interesting work recently reported by Ormerod,[3] who has studied the mode of action of antrycide—the active new remedy for trypanosomiasis in cattle which has raised such great hopes of curbing this economic blight in Africa. After demonstrating that antrycide also belongs to the group of compounds described above, Ormerod shows that it causes the appearance of peculiar inclusion bodies in the cytoplasm of the trypanosomes. These bodies are basophilic and also faintly fluorescent. Similar inclusion bodies are produced by effective doses of suramin and of phenanthridinium compounds. By appropriate stains they are shown to contain ribonucleic acid and protein. Ormerod conceives that antrycide and suramin penetrate slowly into the cell, where they are retained because their molecules carry a permanent electric charge; that their action is chiefly on the cytoplasm and not primarily on the nucleus; and that they inhibit the growth and division of trypanosomes by preventing the normal interaction of protein with ribonucleic acid. Much of this conception is bold speculation rather than established fact. Nevertheless its boldness may well prove a stimulus to further work which will assist in the elucidation of the mysteries of intracellular reactions.

ALCOHOLISM IN BRITAIN

Not so long ago it could be said that alcoholic excess was a serious problem in Britain, affecting large numbers of male wage-earners in industrial towns, accentuating their low living standards, and increasing the misery of their family life. Men drank to escape from a sordid environment and from unbearable psychological conflict. "The quickest way out of Manchester" was to get drunk. Hogarth in his "Beer Street" and "Gin Lane" illustrated the scale of these evils among the London masses of the mid-eighteenth century, but it was in the nineteenth century that their horror reached its peak.

A gradual improvement in living conditions has led to a decline in this type of drinking. The total consumption of beer and spirits has remained high, but it is spread more evenly over the population as a whole. There has been an increase in moderate drinking and a decrease in convictions for drunkenness. Women drink more and men less. The drinking of alcohol has been sanctioned by society, in that it has become a convivial activity of the group, approved by the group and at the same time moderated by it. Where a man would previously have gone out to a pub to escape the squalor of his home, now he takes his wife with him. More than a vestige of the older pattern remains, but historically the trend is towards a breaking of the vicious circle linking drink and poverty.

Social drinking is a central feature in many societies, large and small, simple and complex. It is an essential part, for example, of the civilization of France and Italy. In many religions, including Christianity, the act of drinking wine has a high symbolic value. This pattern persisted in Britain, especially in the countryside and smaller town, through the period of social disintegration due to industrialism. The tradition has remained strong enough to make the present-day public-house a centre of the kindlier and more genial side of our social nature. Already in Shakespeare's time there were puritans who condemned drinking out-and-out, and Falstaff is eloquently scornful of them: "Nor a man cannot make him laugh; but that's no marvel, he drinks no wine."

Yet it is in the regions most dominated by the puritan ethic that alcoholic excess appears most pronounced. Where the social group withdraws its approval from drinking, it becomes either a solitary vice or a wickedness covertly shared with a few boon companions. This type of alcoholism is allied not so much to poverty as to conflict within the personality. It is to be found in countries such as the U.S.A. and Sweden, which have experimented in prohibition. These two countries head the list given in the extremely interesting and valuable first report on alcoholism issued by the World Health Organization[1] as having the highest proportional number of alcoholics—Italy, that great wine-drinking country, having the lowest.

It should be added that both the U.S.A. and Sweden have tackled what is for them a serious social problem with outstanding vigour and insight. The American society calling itself "Alcoholics Anonymous" has achieved a remarkable success by restricting its membership to alcoholics who have been able to become, or are attempting to become, abstinent. They thus, in a sense, bring back into society the individual who has become cut off from it by addiction: "The alcoholic's best hope of remaining abstinent lies in helping others to achieve that state."

The W.H.O. Report gives a provisional figure (1,100 per 100,000 adults) for the proportional number of alcoholics in England and Wales, which is barely a quarter of the American figure. Enough of Sir John Falstaff may have lingered in our midst to mitigate the severity of the Nonconformist conscience and its characteristic personality conflicts. None the less, every society has its alcoholics in all classes, and their unhappy condition is one which the social scientist as well as the medical profession must try to understand.

URINE CHLORIDE ESTIMATIONS

Salt deficiency is an important possible result of vomiting, diarrhoea, or sweating from any cause, and is often sufficiently severe to threaten life if unrelieved. The syndrome has been increasingly recognized since the work of Gamble and his co-workers[1 2] and of Marriott,[3] who have described in detail the plasma electrolyte changes which occur and have revolutionized the treatment of the condition.

[1] The Expert Committee on Mental Health. Alcoholism Subcommittee. Report on the First Session, December 11–16, 1950.

[1] *J. clin. Invest.*, 1925, **1**, 403.
[2] *Chemical Anatomy, Physiology and Pathology of Extracellular Fluid*, 1947, Harvard University Press.
[3] *British Medical Journal*, 1947, **1**, 245, 285, 328.
[4] *J. Amer. med. Ass.*, 1936, **107**, 14.

The Fantus[3] method of urine chloride estimation has played an important part in the development of this subject and has appeared to provide an easy short-cut to the diagnosis of salt deficiency. The method is, in fact, so deceptively easy that its limitations are not perhaps generally appreciated. Some of these are dealt with in detail in Dr. W. H. Taylor's article in this issue (p. 1125), which should be read with care by all who wish to interpret the results of this test at all critically.

The difficulties which arise are of two types. First, the estimation itself is liable to considerable experimental error, particularly when carried out hurriedly in the ward side-room. Secondly, in pathological states the chloride excretion is by no means a safe guide to the body requirements. Errors can appear in both directions: patients with high plasma chloride levels may at times fail to excrete chloride, whereas other patients with salt deficiency may continue to excrete considerable amounts of the ion. This is particularly true of subjects in whom salt deficiency is complicated by disturbances of acid-base balance.

The fact is that an estimation of this type can never hope to do more than give a rough prediction of the level of plasma chloride, and is therefore bound to be less exact than the plasma chloride estimation itself. Furthermore, it will be successful in predicting this level only if the method of estimation is satisfactory and if, in addition, renal function is reasonably normal, particularly the renal threshold for chloride. This threshold depends mainly on the adrenal cortical control of the tubular reabsorption of electrolytes, but can also be disturbed by gross acidosis or alkalosis. It follows therefore that, while the method may be perfectly satisfactory, as shown by Marriott,[3] in the study of salt deficiency in healthy adults under tropical conditions, the reverse may be true in at least some patients with poor renal and adrenal function.

Finally, even supposing that the plasma chloride level could be successfully predicted by a test for urine chlorides, this still does not tell the whole story about the body's need for sodium and chlorine, for this depends also upon alterations in plasma and extracellular fluid volumes. Because of the possible fallacies, therefore, this apparently simple test should not be used as the sole guide to electrolyte therapy.

BILATERAL POLYCYSTIC OVARIES

In 1935 Stein and Leventhal first reported a syndrome, to which their names are sometimes appended, associated with bilateral polycystic ovaries in women usually between 18 and 28 years of age.[1] The most constant features of the clinical picture are secondary amenorrhœa, sterility, and a tendency to the masculine type of hirsuties. Underdevelopment of the breasts, obesity, and acne are less often present. The characteristic physical sign is a firm, smooth, non-tender enlargement of both ovaries to three or four times normal size. This enlargement may be detected only at examination under anaesthesia, especially in obese women, but combined pneumoperitoneography and hysterosalpingography can be used to give a clear definition of the ovaries. Since the original description appeared Stein and his associates,[2-4] as well as others[5 6] in the United States, have published further reports, while in this country a similar condition was described by Bailey[7] in 1937.

This syndrome, although imputed to a disturbance of the anterior-pituitary–ovarian cycle, has proved refractory to treatment with hormones, with the possible exception of thyroid extract, but good results have been obtained by wedge resection of both ovaries, a procedure practised by Stein as early as 1929. This form of surgery has been followed by a return of normal menstruation in a proportion of cases varying from 68%[5] to 90%,[4] and in nearly as many cases the associated sterility is also said to have been relieved. Moreover, hirsuties has been improved or arrested in its progress, though never disappearing. Very few relapses have been seen after operation, and in some patients subjected to laparotomy in later years the ovaries have looked normal.

While the results of this form of treatment in carefully selected patients are reported as excellent, the *modus operandi* of such surgery and the pathogenesis of the ovarian condition remain obscure. The latter problem has recently been discussed by Leventhal and Cohen.[8] It has generally been held that the basic disorder lies in some hormonal imbalance between the anterior pituitary and the ovaries, yet the secretion of follicle-stimulating hormone was found to be within the normal range in 12 patients.[5] The occasional association of multiple follicular cysts in the ovaries and adrenal cortical hyperplasia is perhaps relevant to this discussion. On the other hand, the researches of Reynolds[9] and Delson[10] suggest that changes in the ovarian blood vessels play an important part in the formation of the cysts, the fluid content of which appears to be a transudate. These vascular changes, however, might be secondary to some psychological or hormonal disturbance rather than primary. Leventhal and Cohen state that the characteristic histological features of the ovaries are a thick fibrous tunica albuginea, multiple follicular cysts with perifollicular fibrosis, and, most consistently, a marked hyperplasia of the theca interna cells with numerous mitoses. The theca cells sometimes show luteinization, but the absence of corpora lutea is striking. These changes support the suggestion of Ingersoll and McDermott[5] that the theca interna cells produce hormones, and indeed there is not only experimental but also clinical evidence that these cells can produce androgenic as well as oestrogenic substances,[11] thus accounting for the defeminization or degree of masculinization seen in a proportion of women with this syndrome. But, while this theory may help to explain certain variations in the clinical picture, the basic cause remains unknown, and the success attending wedge resection of the ovaries remains unexplained.

1 *Amer. J. Obstet. Gynec.*, 1935, **29**, 181.
2 Ibid., 1939, **38**, 465.
3 Ibid., 1945, **50**, 385.
4 Ibid., 1949, **58**, 267.
5 Ingersoll, F. M., and McDermott, W. V., ibid., 1950, **60**, 117.
6 Meaker, S. R., *Fertility and Sterility*, 1950, **1**, 293.
7 *J. Obstet. Gynaec., Brit. Emp.*, 1937, **44**, 637.
8 *Amer. J. Obstet. Gynec.*, 1951, **61**, 1034.
9 *Endocrinology*, 1947, **40**, 388.
10 *Amer. J. Obstet. Gynec.*, 1949, **57**, 1120.
11 Culiner, A., and Shippel, S., *J. Obstet. Gynaec., Brit. Emp.*, 1949, **56**, 439.

DISK PROTRUSIONS IN THE DORSAL SPINE

Protrusions of the intervertebral disks in the dorsal region of the spine cause symptoms much less often than cervical and lumbo-sacral disk protrusions: among 5,500 operations for protruded disks performed at the Mayo Clinic between 1939 and 1945 only 12 were for lesions in the dorsal region.[1] Little attention has been given to lateral disk protrusions as a possible cause of pain in the chest or abdomen, for the neurological signs of interference with function of a single root in the dorsal region in the form of weakness of one set of intercostals, and possibly minimal sensory impairment, are inconspicuous. As in the other regions of the spine it is probable that lateral protrusions may cause only root symptoms. Midline disk protrusions, however, may cause compression of the dorsal region of the spinal cord, and before Mixter and Barr's[2] fundamental work had drawn attention to the importance of disk protrusions cases had been diagnosed as extradural ventral chondromata[3] and ecchondroses of the intervertebral fibrocartilage.[4] Ragnar Müller[5] has recently described four cases operated on at the Serafimer Hospital in Stockholm. The patients were all men between the ages of 40 and 60, and none gave a history of injury. The presenting symptom was usually unilateral root pain, and this was followed by a progressive transverse lesion of the spinal cord, though in one case there was a marked remission of symptoms for several years. Plain radiographs showed calcification within the damaged disk and narrowing of the disk space in two of the cases; in the other two no abnormality was apparent. Myelography disclosed a complete subarachnoid block in one patient and an incomplete block in the other three, but did not help in elucidating the cause of the block. A correct pre-operative diagnosis was made in only one of the four patients. At operation a calcified disk protrusion was removed in each case, but the results of operation were uniformly bad: the patients developed an almost complete and apparently permanent transverse lesion of the cord, presumably due to damage to the cord during the difficult manipulations necessary to remove the disk protrusion. Müller suggests that better results might be obtained by a simple decompression of the cord without any attempt to remove the protrusion.

GENERAL PRACTITIONERS' PAY

The Annual Conference of Representatives of Local Medical Committees on October 31[6] endorsed the action taken so far by its executive, the General Medical Services Committee, to bring to arbitration the size of the general practitioners' central pool. The Special Conference held on July 19 instructed the Committee to ask for arbitration; in negotiations with the Ministry of Health and the last Minister agreement was obtained that an adjudicator would be appointed for this task. In thus clearing the ground for the next stage—the preparation of the general practitioners' case—the G.M.S. Committee has rooted out several undesirable weeds that had sprouted there. First, the Government has been persuaded that the remuneration to be paid must be studied on its own merits and not be decided by any arbitrary maximum such as the additional £2m. offered in the summer. Acceptance of that offer would have been a surrender to those people who find the Spens Report an embarrassment. Secondly, the attempt to link any additional pay with economy in prescribing has gone. The need for this economy is admitted by everyone, certainly as much by the medical profession as by the Government, though for different reasons. From the doctors' point of view over-prescribing is bad doctoring, but equally so is under-prescribing. As we have pointed out before, a minority of patients exert a thoroughly unpleasant kind of pressure on doctors. There are better ways of effecting economy here than by trying to cut down the cost of the general-practitioner services at the expense of the general practitioners' pay, and the G.M.S. Committee has succeeded in impressing this view on the Ministry.

There has been some give-and-take on the terms of reference to be set before the adjudicator and on those that will guide the Working Party to investigate distribution of the pool. In deciding what should be the present remuneration of general practitioners the adjudicator will be asked to provide a figure that is determined by three main factors. These are the initial Spens figures, the change in the value of money since 1939, and the extent to which incomes in other professions have increased since then. "All other relevant factors" are also to be considered. The Working Party on distribution will meanwhile have started work, but the Ministry has given the important assurance that the adjudicator's award will be published whether or not the Working Party has arrived at its conclusions. The Minister also promised that arbitration on any fundamental disagreements in the Working Party "is not ruled out." This is merely a useful safeguard made at the G.M.S. Committee's request, but in fact every effort will be made to reach agreement.

Having received the approval of the Conference for its work, the Committee now will continue it by preparing the evidence. The Committee has appointed a special Evidence Subcommittee to study the case and to gather evidence. Expert advisers have been chosen to help it, and counsel will soon be briefed to present the case to the adjudicator. The work will be increased by the need to collect evidence relating not only to the present financial circumstances but also to those in the last three years, for the Committee will press for retrospective adjustment of the central pool back to the Appointed Day. Members of the Conference were naturally eager to know when the adjudication is to begin, but they rightly refused to tie down their negotiators to any fixed dates. Preparation of the case and briefing of counsel is bound to take several months if it is to be complete; and of its completeness there must be no question, for the adjudicator's decision will be one of the most important ever made on general practitioners' pay.

[1] Love, J. G., and Kiefer, E. J., *J. Neurosurg.*, 1950, **7**, 62.
[2] *New Engl. J. Med.*, 1934, **211**, 210.
[3] Elsberg, A., *Bull. Neurol. Inst. N.Y.*, 1931, **1**, 350.
[4] Hawk, W. A. *Brain*, 1936, **59**, 204.
[5] *Acta med. scand.*, 1951, **139**, 99.

[6] See *Supplement*, p. 197.

REFRESHER COURSE FOR GENERAL PRACTITIONERS

HAEMOLYTIC DISEASE OF THE NEWBORN

BY

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Few have the opportunity of keeping abreast with the advancing knowledge about haemolytic disease of the newborn. Yet in considering the disease this knowledge must be constantly applied by medical practitioners. It seems reasonable, therefore, that the subject should be reviewed from time to time in a manner which may help the family doctor.

The severe forms of this disease have long been recognized by their clinical patterns as: (1) *Hydrops foetalis* —the oedematous stillborn infant; (2) *icterus gravis neonatorum*—the baby deeply jaundiced within a few hours; and (3) *congenital anaemia*—the baby developing intense pallor within the first two or three weeks of life. These were sometimes collectively known as "erythroblastosis foetalis." The cause remained a mystery until 1940, when Landsteiner and Wiener discovered a new blood-group substance—the Rhesus factor. This substance is present in the red cells of six out of every seven people, and these are therefore said to be Rh-positive, while those lacking the substance are Rh-negative. Should Rh-positive cells enter an Rh-negative person, as can occur when an Rh-positive foetus exists in an Rh-negative mother, then that person may react by producing antibodies able to destroy any Rh-positive cells. These antibodies, crossing the placenta, destroy the foetus's red cells. Since the clinical features —anaemia, jaundice, and oedema—found in many affected babies are mainly due to this haemolysis, the condition is best described as "haemolytic disease of the newborn."

As haemolytic disease affects only one baby in every 200 born, a busy practitioner is likely to meet a case in his own practice only every three to four years, but when he does his management may mean the difference between life and death to the affected infant. Moreover, the subject has been widely presented in the lay press, and, as one in eight marriages will be between an Rh-negative woman and an Rh-positive man, the family doctor is likely to be consulted frequently.

Nomenclature and Diagnostic Tests

By testing the blood it is possible to divide the population into two groups—those whose cells contain Rh substance being Rh-positive, while those lacking this substance are Rh-negative. The Rh group can be further divided into many subgroups. There are about a dozen Rh substances, all antigenic, but only one, D (big D), is of great clinical importance. Therefore as a routine people are tested only against anti-D serum, and confusion is avoided if D-positive or D-negative are used synonymously with Rh-positive or Rh-negative respectively. Throughout this article only the terms "Rh-positive" and "Rh-negative" will be employed, as they are already established in common usage. In order to avoid any misunderstanding many laboratories report results as Rh(D)-positive or Rh(D)-negative, or, alternatively, state the specific type of grouping serum against which the cells have been tested.

A person inherits his Rh group from both parents, so that if the Rh-positive factor is inherited from each he will be *homozygous Rh-positive*. If the Rh-positive factor is inherited from one and Rh-negative from the other, then he will be *heterozygous Rh-positive*. Children of an Rh-negative mother and a homozygous Rh-positive father must themselves be Rh-positive, while those with a heterozygous Rh-positive father have an equal chance of being Rh-positive or Rh-negative, depending on which factor is transmitted. Similarly an Rh-negative father can only hand on to his children the Rh-negative factor.

The Coombs test, best carried out on the infant's cord blood, is used to show whether antibodies have, in fact, crossed the placenta and coated the infant's red cells. Any newborn infant with a positive Coombs test, whether with or without clinical symptoms, must be considered to have haemolytic disease. A negative result excludes the disease. The test often but not necessarily remains positive for the first three to four weeks of life.

Errors, clerical and technical, do occur with all laboratory tests: therefore clinical evidence of disease should always be regarded, and if this is in conflict with such tests they should be repeated. Last year a doctor, when called to see a baby aged 10 days with severe pallor, found that the mother's blood-group card stated that she was Group O and Rh(D)-positive. This caused him to discard the diagnosis of haemolytic disease until the fourteenth day, when the baby was so ill that it died. But the laboratory test was at fault and later tests showed that she was actually Rh-negative.

Aetiology

The stage is set for the disease whenever an Rh-negative mother carries an Rh-positive foetus; a combination of circumstances occurring in one of every ten pregnancies. The disease will not occur in the infant unless Rh antibodies develop in the mother. Such immunization of the mother can occur if she ever receives Rh-positive cells, either by transfusion or intramuscular injection at any time in her life, or from her own foetus during pregnancy. The risks, however, are not equal. The risk of immunization by pregnancy is one in twenty, while transfusion carries a fifty-fifty risk. Thus it is imperative at all times that the transfusion of Rh-negative women and girls must be with Rh-negative blood. If an Rh-negative woman or girl has been immunized by intramuscular or intravenous injection of Rh-positive blood, the first infant, if Rh-positive, may be affected. If immunization occurs through pregnancy, the first infant is usually spared, but the likelihood of disease increases with each succeeding infant.

With this range of possibilities the disease must be kept in mind whenever pregnancy occurs in an Rh-negative woman, and must be expected if Rh antibodies are found in her serum. Once immunization has occurred it is permanent, and every subsequent foetus is at risk.

Common Clinical Patterns

Babies affected with haemolytic disease may show differing pictures, varying from the baby without clinical evidence of disease to the hydropic stillborn foetus. Cases may fall anywhere between these extremes, but in general we recognize four fairly distinct types.

1. Subclinical Variety

This is the Coombs-positive baby in whom the diagnosis cannot be made on clinical grounds, and who requires no active treatment. Such cases are nearly always detected because antibodies have been found in the mother's serum before delivery and therefore the Coombs test has been done on the baby's cord blood at birth. Some babies in this group may have transient jaundice indistinguishable from physiological jaundice of the newborn, and anaemia, if present, is minimal, of short duration, and does not require active treatment.

2. Anaemic Variety

This is the baby in whom pallor is the striking feature. Anaemia usually becomes evident towards the end of the first week and increases thereafter. If not recognized early, by careful attention to the colour of the mucous membranes, pallor may only be noticed as late as three to four weeks, when the haemoglobin level can be as low as 20% and the baby's life is in danger. Jaundice often occurs in these infants, but, although frequently of earlier onset and perhaps of greater depth than the physiological variety, it is in no way diagnostic, nor is it essential for the diagnosis. Pallor in a baby may be easily overlooked unless the eye be trained to recognize it. Moreover, if jaundice is present, pallor may be obscured and only become evident when jaundice fades. One evening a doctor telephoned to say that an 8-day-old baby had "rhesus disease." In answer to questioning we learnt that the baby seemed lively and took its feeds well. The baby, he said, "was quite a deep orange colour a few days ago, but is much better now—it is only a pale lemon colour." We should have been alarmed rather than reassured by that description, for that baby while being brought for examination the next morning died from anaemia in its father's arms in the hospital lift.

Even now there is a considerable mortality in this anaemic type of disease which could by careful supervision of each baby be reduced to less than 1%. In the first six weeks, in addition to assessing the general condition of the baby and observing the colour of mucous membranes, haemoglobin estimations should be made every two to three days (and, if low, even more frequently). No elaborate method is needed, and the "Tallqvist" method* is accurate enough at levels below 50%. With the aid of this simple test, no child need die solely because anaemia has not been recognized.

Recently we have seen two babies who owe their lives to such careful observation. The first was a baby in a small maternity hospital who at the age of 5 days was noticed to be pale. As treatment entailed a night journey of 60 miles (96.5 km.) it was suggested that a haemoglobin estimation be made. This was done by a complex method and the result was 65%, but the matron insisted that the baby looked much paler than this and that she proposed to bring it to hospital nevertheless. On arrival three hours later the haemoglobin was 35%; the baby was transfused and recovered. The second was a baby, said to be Coombs-negative and therefore unaffected, discharged from a maternity hospital. In the face of this, the family doctor recognized the occurrence of abnormal pallor at four weeks and sent the baby back to hospital. It was found that the report of a negative Coombs test had been sent in error, and this baby also survived.

*See *British Medical Journal*, 1951, **1**, 1093.

3. Deeply Jaundiced Variety

This is the baby with early rapidly deepening jaundice and early progressive anaemia. Many babies in this group have enlarged liver and spleen. Some have small round clearly defined bruises of the skin, especially on the face, but sometimes also on the trunk and limbs. The jaundice may obscure pallor, which is difficult enough to recognize in a newborn baby in any case. Yet the degree of anaemia at birth is often closely related to the outlook for the baby; hence in hospital it is customary to do a haemoglobin estimation on the baby's cord blood. A level of less than 50% invariably means severe disease with a very high mortality rate, whereas levels of more than 100% are frequently associated with mild disease having a low mortality rate.

The mortality in this, the deeply jaundiced variety of haemolytic disease, is high, death usually being due to: *(a)* heart failure on the first day in babies grossly anaemic at birth; *(b)* brain damage during the first week, usually in deeply jaundiced babies; or *(c)* haemorrhages into internal organs.

3a. Brain Damage (Kernicterus)

This complication is almost completely confined to deeply jaundiced babies and is therefore best considered at this point. Of all liveborn babies with this illness, one in twelve suffers brain damage, the diagnosis usually being evident in the first week of life.

Neurological diagnosis in the newborn is notoriously difficult, but one should consider the possibility of kernicterus if a deeply jaundiced baby with haemolytic disease suddenly, at about the third or fourth day, develops head retraction. Other features often develop, particularly extensor spasms of the arms and legs with clenching of the fists. A shrill cry, vomiting, and fits complete the picture. Once kernicterus has occurred, treatment is of no avail and death from respiratory failure usually follows in a matter of days. The few that survive have permanent brain involvement, which is discussed later.

It is not uncommon for any jaundiced baby to be drowsy and to take feeds badly. This in itself does not denote kernicterus, but if neck stiffness develops it is important to obtain specialist opinion. To distinguish kernicterus from cerebral birth injury and other cerebral disease will require expert examination by a doctor experienced in the care of the newborn child.

4. Stillborn Variety

The most severe form of haemolytic disease kills the foetus. The stillbirth due to haemolytic disease often presents a peculiar appearance. It is grossly oedematous, with protruding tongue, waterlogged tissues, and free fluid in all the body cavities. Pallor is extreme and the liver and spleen are greatly enlarged. Very occasionally a grossly oedematous baby is liveborn but dies in a matter of minutes. We must not oversimplify the position, and it must be realized that oedematous stillbirths may be caused by diseases other than haemolytic disease. Nor are all haemolytic disease stillbirths oedematous; many are indistinguishable

superficially from stillbirth due to other causes. Of course a foetus with haemolytic disease may be stillborn owing to causes other than the disease itself.

Essentials of Treatment

The first essential is early diagnosis. All cases should be anticipated by blood-grouping tests carried out before delivery. The routine antenatal blood-grouping tests are detailed later, but if antibodies are detected haemolytic disease is to be expected.

Every mother whose serum contained antibodies should be delivered in a hospital specially prepared to treat haemolytic disease of the newborn, which is a difficult technical procedure often requiring early exchange transfusion. Furthermore, as there is a high incidence of post-partum haemorrhage in the mothers of these babies, it is desirable that they, too, should have facilities for early treatment with Rh-negative blood, if needed.

Once antibodies have developed they cannot be nullified, nor, within the limitations of our present knowledge, can the foetus be protected from their action.

Premature Induction of Labour.—There is evidence that premature induction does not improve the baby's chance of survival, and therefore we ourselves have not used this form of treatment. Moreover, nothing could be more tragic than that an Rh-negative baby, and therefore an unaffected one, should be delivered before term and die because of prematurity. There may be a case for premature induction in families in which there is a previous history of stillbirth due to *haemolytic disease*, but only if the father is homozygous Rh-positive, meaning that all his children must of necessity be Rh-positive and therefore affected.

Blood Transfusion.—Few would question the value of simple transfusion for the relief of anaemia during the course of the illness. Recently, early exchange transfusion in babies with severe disease has held out some hope of a reduction in mortality, and also in the prevention of brain damage. These are hospital procedures, and if the baby is to be given the best chance it must be delivered in hospital.

Breast-feeding.—It has been suggested that breast-feeding may be harmful in this disease, by transferring additional antibodies from the mother to the baby. We encourage breast-feeding in all cases of this disease, and prolonged haemolysis due to this cause is extremely rare.

Contribution of the Family Doctor

The family doctor shares with the midwife and the staffs of antenatal clinics the responsibility of antenatal diagnosis of this disease. He must sometimes also consider the diagnosis in families in which full blood-group studies have not been made and in which the first indication of trouble may be the appearance of suggestive symptoms in the baby.

Clinical evidence of haemolytic disease should never be disregarded, and, when there is doubt, further opinion should be obtained even if the parents' blood groups are known and are not the classical "set up" of Rh-negative mother and Rh-positive father.

As babies with this illness must be kept under observation until the age of 6 weeks, the hospital will probably wish the family doctor to share in this. Hence it is desirable that he should be familiar with the possible courses of this illness. He should also seek to protect his patients from the advice so many ill-informed people seem anxious to give, and should make it his responsibility to make sure that they receive only the best advice available.

The responsibility of the family doctor is considered under the following headings: *(a)* to detect families in which haemolytic disease will probably occur; *(b)* to diagnose the disease in families in which blood-grouping tests have not been carried out; and *(c)* to advise the Rh-negative person.

(a) To Detect Families in which Haemolytic Disease will Probably Occur

This is done by the following routine serological tests on every expectant mother before delivery (see Diagram).

1. *Determine the Rh Group of the Mother at her First Attendance.*—If Rh-positive (six out of seven women) there is no further need for tests in the absence of clinical disease in the infant. If Rh-negative the mother may become immunized by pregnancy if her husband is Rh-positive, therefore:

2. *Determine the Rh Group of the Husband.*—If Rh-negative there is no need for further tests in the absence of clinical disease in the infant, and the mother can be told that there is no risk. *If Rh-positive* the mother should be told that it is a twenty-to-one chance against anything going wrong, but that tests on her blood must be made between the thirty-second and the thirty-sixth week of each pregnancy. If the father's blood is not available for grouping he should be assumed to be Rh-positive.

3. *Test the Mother's Serum for Antibodies Between the Thirty-second and Thirty-sixth Week of Every Pregnancy.*—If antibodies are not present no disease of the infant should be anticipated and the pregnancy should be treated as normal. However, a specimen of *cord blood* from the baby at birth should be obtained for the Coombs test and Rh grouping, as these will detect haemolytic disease even if antibodies were not detected because of technical error or failure to collect the specimen. If the baby is Rh-negative and the father is known to be Rh-positive, it will establish that he is heterozygous. *If antibodies are present* the baby will, if Rh-positive, suffer from some form of haemolytic disease, and arrangements should be made for its delivery in hospital. Every now and again, under these circumstances, the baby will be Rh-negative, the same as the mother, and therefore unaffected. One cannot predict accurately the group of the baby before birth, but some indication can be obtained by genotyping the father.

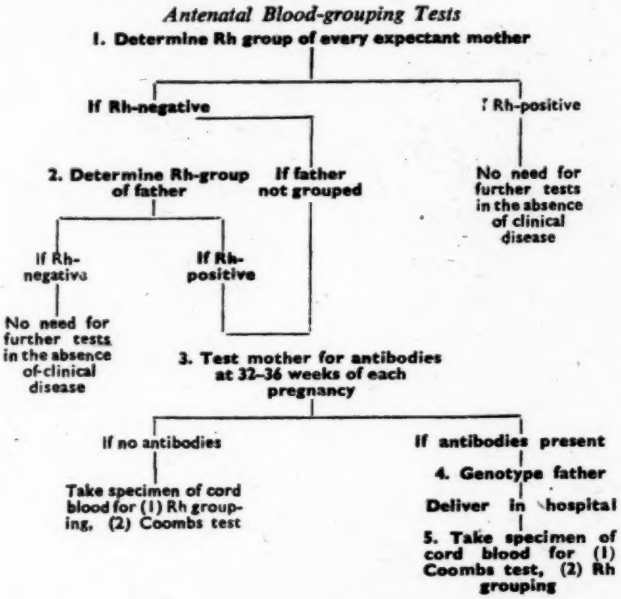
4. *Determine the Genotype of the Father.*— If homozygous, the doctor is in a position to tell the parents that the baby is certain to have some form of haemolytic disease; if heterozygous, that there is a 50% chance that it will be Rh-negative and therefore normal. Although of limited value when a woman is pregnant, the knowledge of the genotype will often enable parents to face the question of further children more intelligently.

The previous history of one of our patients may be of interest. Her first pregnancy ended in abortion, and blood transfusion was necessary to save her life. She was given 4 pints (2.3 litres) of blood, the Rh group of which was unknown. The next pregnancy resulted in a baby who became severely anaemic during the second week of life,

and who was admitted to hospital but died of anaemia. The next two pregnancies resulted in stillborn hydropic foetuses. At this stage her husband was genotyped and found to be heterozygous Rh-positive. This gave her a fifty-fifty chance of a normal baby, and this year she was delivered of a healthy Rh-negative daughter.

5. *Cord Blood must be Collected from the Babies of all Mothers with Antibodies.*—The specimen is used for Rh grouping and the Coombs test. If Coombs-positive the diagnosis of haemolytic disease is established, if Coombs-negative the diagnosis is excluded and the baby is presumably Rh-negative.

Antenatal Blood-grouping Tests


1. Determine Rh group of every expectant mother
If Rh-negative
If Rh-positive
2. Determine Rh-group of father
If father not grouped
No need for further tests in the absence of clinical disease
If Rh-negative
If Rh-positive
No need for further tests in the absence of clinical disease
3. Test mother for antibodies at 32–36 weeks of each pregnancy
If no antibodies
If antibodies present
Take specimen of cord blood for (1) Rh grouping, (2) Coombs test
4. Genotype father
Deliver in hospital
5. Take specimen of cord blood for (1) Coombs test, (2) Rh grouping

To summarize: By means of blood-grouping tests it is now possible to determine which mothers are at risk, to anticipate disease in nearly all cases, and to establish the diagnosis within a few minutes of birth. Tests for the strength of antibodies in the mother's blood are not of much value in predicting the form or severity of the disease in the baby, and are not required. The determination of the father's genotype will often help parents to face the future more confidently, and should be suggested in any family in which the mother has antibodies.

(b) To Diagnose the Disease in Families in which Blood-grouping Tests have Not Been Carried Out

A practitioner called to see a baby who is jaundiced or pale in the first few weeks after birth should always consider the possibility of haemolytic disease. The clinical picture may be so striking that the diagnosis is certain, but in other cases it may be impossible to make a definite diagnosis without recourse to serological tests.

If a baby is heralded by yellow liquor, is pale at birth, and yellow within eight hours, with enlargement of liver and spleen, the diagnosis is sure and the disease is severe. In addition, should there be small round well-defined bruises of the face, trunk, and legs, or frank haemorrhage from any mucous membrane, or if oedema is present, treatment is urgent.

Jaundice in the first 24 hours (best seen over the abdominal wall with the skin stretched) is due to haemolytic disease until it is proved otherwise. Proof of diagnosis can only readily be obtained by serological investigation, and this should always be carried out. While of such immense diagnostic importance, early jaundice does not invariably mean severe disease. A history of clinical disease suggesting haemolytic disease of the newborn in previous children may be helpful in some cases, but is by no means always present.

Anaemia occurring in the neonatal period is most likely to be due to haemolytic disease. Oedema, if severe, is a sign of impending death. Haemorrhage, apart from petechiae on the trunk or face, gives cause for alarm. Anaemia is of prognostic value, while jaundice is that which focuses attention.

(c) To Advise the Rh-negative Person

To answer many of the questions asked by Rh-negative women is often difficult because no single test or combination of tests gives a precise indication of the outlook. However, by the simple expedient of grouping the husband's blood, one in seven will be found to have Rh-negative husbands and can therefore be told that there is virtually no risk. Those with Rh-positive husbands can be told that there is only a one-in-twenty chance of any trouble. If antibodies are not present at 32 to 36 weeks the mother can be told that the baby will not have haemolytic disease. If, however, antibodies are detected the mother must be told that the child may have haemolytic disease, and that she should be delivered in a hospital prepared to treat this disease. Serological tests, unfortunately, are of little value in predicting the form or severity that the disease is likely to take in the baby, and we have found the previous history of only limited value.

The first baby is unlikely to have haemolytic disease unless the mother has been exposed to the risk of immunization by previous blood transfusion, but all are familiar with the difficulty of being certain that any pregnancy is indeed the first. In general, the first affected baby is but mildly affected and recovers completely. Subsequent babies are likely to have increasing degrees of severity, and, in particular, the occurrence of a stillbirth due to haemolytic disease of the newborn will increase the chance of subsequent pregnancies ending in this way. The sequence of progressive severity is happily not invariable, so that a mildly affected baby may follow one severely affected, dying with brain damage, or even stillborn. The converse is also true—namely, that the first affected baby may be severely affected or even stillborn. Some families tend to produce identical disease in all children, as in one family in which three affected babies have survived after transfusion for relief of anaemia during the second week of life.

The probable form that haemolytic disease took in the earlier children can be assessed only by questioning on such points as stillbirth, jaundice, anaemia, etc. If previous children have died, then information concerning age and mode of death may be helpful in the assessment.

Finally, our evidence suggests that immunization produced by transfusion is of the same significance as that caused by pregnancy.

Outlook for Affected Infants

I have had the opportunity of studying 600 cases of this disease in recent years. Approximately 15% were

stillborn. Of those born alive, approximately 15% died. Included in these there are some deaths that I believe could have been avoided.

Of all liveborn babies with this illness, one in twelve suffers brain damage, the diagnosis usually being evident in the first week of life. Most of these children die, if not in the first week, then frequently in the first month. A few children with evidence of brain involvement do survive, and such damage may lead to deafness, speech defect, mental defect, or motor disturbance of choreo-athetoid type. Survival is exceptional, however. In fact, 99% of our surviving cases show no gross abnormality. Occasionally teeth of the first dentition may be green, but are otherwise healthy and greatly prized by patient and dentist alike. The permanent teeth are never affected. I know of no other condition definitely attributable to haemolytic disease.

Summary

Haemolytic disease of the newborn may occur whenever an Rh-negative woman carries an Rh-positive foetus.

The disease can be anticipated in almost every case by the family doctor knowing the Rh grouping of the parents in his practice, and seeing that the pregnant Rh-negative mother is tested for antibodies without fail between the thirty-second and thirty-sixth weeks of pregnancy.

If antibodies are present clinical disease can be expected in the infant, and although the severity may vary it is best for such mothers to be delivered in hospitals prepared to treat this illness.

This means that transfusion with Rh-negative blood can be given by the direct or exchange methods without delay; the infant should be carefully watched during the dangerous first week of life.

Should the birth take place at home, a specimen of cord blood should always be taken.

Jaundice in the first 24 hours is due to haemolytic disease until proved otherwise.

After this, when the infant returns home, or in children born at home, the doctor must be on the look-out for delayed anaemia until the sixth week.

In view of the dangerous sensitivity which follows the transfusion of Rh-negative women with Rh-positive blood, all women known to be Rh-negative or women whose Rh group is unknown should be transfused only with Rh-negative blood.

Although immunization through pregnancy cannot be avoided, and immunization, once present, persists for life, a knowledge of the father's complete Rh type and the previous obstetric history will help the family doctor to give some advice about future pregnancies.

If the results of laboratory tests conflict with the clinical picture, such tests should be repeated.

Finally, I would counsel doctors to be careful not to cause unnecessary alarm. To give a gloomy prognosis to all Rh-negative women is a heedless procedure. For, although tragedies occur all too often to women who greatly desire a family, they are outweighed by the unexpected successes, many of which are not yet to be explained by the modern understanding of this disease.

In *Nature* (October 13, p. 651) Dr. R. W. G. Wyckoff publishes electron microscope photographs showing influenza virus particles apparently growing out of infected cells of the chick embryo. That is to say, diseased cells are seen in section to have many small cytoplasmic protrusions from their borders, and these protrusions break off as spheres of the same size and shape as influenza virus particles. Further, these phenomena are not observed in healthy tissues, but only after infection with influenza or Newcastle disease. The work is important for the light it throws on the nature of viruses.

DAME HILDA LLOYD: PRESENTATION PORTRAIT

The 34th annual general meeting of the Medical Women's Federation concluded on November 2 with a presentation of her portrait to Dame Hilda Lloyd. The portrait, which is the work of Mr. Anthony Devas, depicts her in her robes as president of the Royal College of Obstetricians and Gynaecologists.

The President of the Federation, Dr. Doris Odlum, in making the presentation before a large audience of women

doctors at B.M.A. House, London, spoke of Dame Hilda Lloyd's eminence in her profession and of the warm affection in which she was held by all her colleagues. She had had an outstanding career in her native city of Birmingham as consultant at the Women's and Maternity Hospitals and as professor of gynaecology and obstetrics at the United Birmingham Hospitals and the University. Women doctors had great pride in the high office to which she had attained in the Royal College—the first woman to hold such an office in any of the Royal Colleges.

Dame Hilda Lloyd said that this occasion gave her the greatest pleasure she had known in her professional life. She had qualified the year before the Federation was established, and she became a founder member under the stimulus of Dr. Mary Sturge, of Birmingham, who followed Dr. Jane Walker as second president. She recalled the names of some of those associated with the earlier days of the Federation—Lady Barrett, Florence Ivens, Christine Murrell, Violet Kelynack, Catherine Chisholm, Clara Stewart, Louisa Martindale, among others. It was sometimes said that the Federation was superfluous because the British Medical Association treated men and women doctors on equal terms. Nevertheless, the Federation was the body to which the Association turned for the views of medical women. The days were not long past when equal opportunities for medical women could not be taken for granted but had

to be fought for. Women were, and probably would always be, in a minority in the profession, and it was necessary that the minority view should be put forward from time to time. She was proud of her long connexion with the Federation and its own fine record. She also hoped the time was not far distant when the other Royal Colleges might elect a woman to their presidencies.

MANAGEMENT OF RHEUMATOID ARTHRITIS

The annual meeting of the British Orthopaedic Association was held in Edinburgh from October 25 to 27, under the presidency of Mr. S. L. Higgs. The first afternoon was devoted to a symposium on the "Management of Rheumatoid Arthritis."

Mr. S. L. HIGGS, introducing the first speaker, said that he had felt concern over the lack of co-operation between physicians and surgeons in the treatment of patients with rheumatoid arthritis. It was common to see patients in medical wards with established deformities which could have been prevented by early orthopaedic care. Physicians often failed to realize what could be done to prevent deformity; and orthopaedic surgeons did not always appreciate sufficiently the help that could be given by their medical colleagues. It was for these reasons that he had suggested this symposium, which he hoped would foster a spirit of closer co-operation in the future.

Rest for the Joints

Dr. J. R. DUTHIE (Edinburgh) said that the introduction of cortisone and adrenocorticotrophic hormone (A.C.T.H.) had tended to obscure other advances that had been made, but these were nevertheless important. A few years ago the hypothesis of focal sepsis as an important aetiological factor was widely accepted. The disease was regarded as essentially a constitutional illness, and it was believed that treatment should be directed primarily towards encouraging constitutional improvement. A wide variety of illogical methods of treatment were suggested and practised, but too little attention was paid to the condition of the joints themselves, which, though inflamed, were subjected to daily passive movements. One of the most important recent advances had been the abandonment of this "uncontrolled empiricism" in the management of the disease. Controlled investigations were now being carried out, and they had already led to the discarding of many remedies for which extravagant and unfounded claims had been made. The only remedy that had survived was gold, and its reputation had suffered to some extent.

It was possible, he submitted, that the constitutional disturbance of rheumatoid arthritis was secondary to the severe inflammatory changes in the joints, rather than the reverse. With this possibility in mind he was investigating the results of resting the affected joints rather than submitting them to passive movements. The limb was rested in a plaster splint and the muscles were exercised by static contractions. Pain was rapidly relieved in most cases, and the need for analgesics was reduced. The inflammation of the joints appeared to settle relatively quickly, and ankylosis had not occurred. After the period of rest, which lasted a few weeks, an attempt was made by resistance exercises to restore joint stability before weight-bearing was allowed.

A notable advance in the approach to the problem of rheumatoid arthritis had been the establishment of special units in which full co-operation between different specialists was possible. Segregation of patients in centres such as these was often criticized, but it had the great advantages for the patient that it facilitated efficient treatment and adequate follow-up examination.

Limitations of Hormone Therapy

Referring to cortisone and A.C.T.H., Dr. Duthie said that the high hopes at first entertained for these substances had not been realized. Though dramatic immediate effects were often obtained, it was clear that an effective cure for rheumatoid arthritis had not been found. Prolonged administration of cortisone or A.C.T.H. was followed by undesirable side-effects in about half the patients treated; yet if the drug was discontinued or reduced relapse occurred. However, in some patients—usually those with mild disease—symptoms could be controlled by long-term administration of the drug in small doses. Research was continuing into the effectiveness of new products—notably substance F—and into the use of cortisone and A.C.T.H. in combination with other drugs, such as *para*-aminobenzoic acid. But real progress must await a better understanding of the fundamental cause of the disease.

"Lively" Splints

Mr. NORMAN CAPENER (Exeter) emphasized the importance of affording physiological rest—or physiological control, as he preferred to call it—to a joint affected by inflammation, whether it be the result of rheumatoid arthritis, pyogenic infection, or other cause. By physiological control he meant rest with the appropriate amount of function applied physiologically, not complete immobilization. He emphasized that the need for physiological control had not been eliminated by the introduction of antibiotic drugs, cortisone, and A.C.T.H.

To put the principle of physiological control into practice he had developed the idea of "liveliness" in splint construction. Instead of providing rigid fixation, splints constructed on this principle allowed a limited amount of controlled movement, yet held the part in the optimum position between the movements. The chief mechanical force in his "lively" splints was spring wire, used whenever possible as an integral part of the construction or framework of the splint. A range of splints employing the principle of "liveliness" was exhibited.

Minor Orthopaedic Measures

Mr. JOHN BASTOW (Bath) described orthopaedic adjuncts to medical treatment, which fell into five groups:

Splints.—These were useful as a protection for inflamed joints in the active stage of the disease, and helped to prevent deformity. Care should be taken to maintain affected joints in the best position. Shells were preferable to complete plasters.

Supports.—These were helpful to protect the weakened joints during remissions of the disease and offered considerable scope to the ingenuity of the appliance maker.

Palliative Operative Procedures.—These included procaine nerve-block, neurectomy, intra-articular injections, arthrotomy and lavage, and bone-drilling. Procaine nerve-block was often useful, but neurectomy was disappointing, because it seemed impossible by a simple operation completely to denervate a joint. Intra-articular injections were not of value in active rheumatoid arthritis, but they might be in the secondary osteoarthritic stage. Arthrotomy and lavage was a useful procedure in cases of persistent effusion; fibrinous masses were removed and the joint was rested in plaster for a few weeks after the operation.

Bone-drilling.—This was at one time used extensively; but it should be confined to cases in which secondary osteoarthritis was associated with extensive sclerosis beneath the joint surfaces. Drilling of the dense bone often relieved the nocturnal pain which was a common feature of such cases.

Joint Manipulation.—This had its uses both in the active stage and in the chronic stage of the disease. In the active stage it should be used only to overcome existing deformity, and should be followed by splinting in the correct position. In the chronic stage manipulation might be used also to improve the range of movement in selected cases. It should be followed by active exercises designed to improve power by progressive increase of resistance. The Guthrie-Smith sling exercise apparatus had proved very useful.

What Surgery Can Offer

Mr. ALEXANDER LAW (London) said that it was often difficult to decide at what stage of the disease orthopaedic operations might prove most useful. He believed, with Smith-Petersen, that surgery had often been left too late and that in some cases it could be advised at a relatively early stage—that was, when medical treatment was beginning to fail. Operations that might be done early included excision of the acromion for disabling subacromial bursitis; excision of the head of the radius in cases in which spasm of the biceps pulled the diseased radial head up against the humerus; and synovectomy in cases of recurrent effusions with extensive synovial disease in the knee-joint.

A second group of operations was applicable only when the active stage of the disease had passed. In the elbow, excision-arthroplasty had a definite place. For arthritis of the wrist with ulnar deviation of the hand he advised excision of the lower end of the ulna with arthrodesis of the radio-carpal joint; this gave a much improved position of grasp. For old disease of the knee with extensive destruction of cartilage arthrodesis was still the best treatment. Attempts to form a useful and painless arthroplasty at the knee had so far met with little success. Severe flexion deformity of the spine could be corrected by "osteotomy" in the lumbar region, whether the deformity resulted from rheumatoid arthritis or ankylosing spondylitis. In the hips, mould arthroplasty as developed by Smith-Petersen gave encouraging results, although movement and function were both restored less completely than was possible in osteoarthritis. For severe affection of the forefeet amputation of the toes was of established value.

In the discussion, Mr. W. D. COLTART (London) emphasized the enormous difficulties that attended the treatment of rheumatoid arthritis. In some respects they were greater than those in tuberculosis or poliomyelitis. Rheumatoid arthritis often went on for as long as 20 years, and this created serious social and economic problems in addition to the clinical problems. He felt that so far as the surgery of rheumatoid arthritis was concerned there were only three operations of established permanent merit—namely, arthrodesis of the knee, arthroplasty of the elbow, and amputation of the toes; in appropriate cases these were of great value. Speaking of training and research, he said that rheumatologists should have an adequate training in orthopaedics, and orthopaedic surgeons should be prepared to help in the training of rheumatologists.

NEW WORK IN RHEUMATOLOGY

The second European Congress of Rheumatology was held in Barcelona from September 24 to 27, under the presidency of Professor A. Pedro Pons. It was attended by over 700 doctors, representing 28 nations. The British delegation numbered 30 and was led by Dr. W. S. C. Copeman. Besides a full academic programme, the Sociedad Española de Reumatismo organized a social programme for the delegates designed to illustrate the various facets of national culture. The programme included displays of Spanish folk dancing, a concert of contemporary Spanish music, a bull-fight, and a banquet in a historic hall, which had last been used for this purpose 450 years ago in honour of Christopher Columbus on his return from his voyage to the New World.

Five main subjects were selected for discussion, each being introduced by two formal papers followed by shorter reports and discussions.

Spondylitis Ankylopoietica

Dr. J. FORESTIER (France) analysed the onset and evolution of spondylitis in his series of 200 cases. In 21% joint symptoms were preceded by iritis or involvement of tendons. A further 21% started with peripheral joint involvement, and only 41% presented with spinal symptoms. In all except six of these the disease started in the lower spine and worked upwards. Radiological changes in the sacro-iliac joints were the only reliable diagnostic aids; there was some evidence that early changes might be reversible. A provocative and closely argued case for considering rheumatoid arthritis and spondylitis as separate diseases was put forward, but unfortunately this implied challenge to current American teaching was unanswered.

Dr. H. VAN SWAY (Holland) pointed out how few satisfactory studies had been made on the pathology of the disease. He described necropsy examinations he had made on three patients, all of whom had died of myeloid leukaemia, which he suggested might have been a complication of the *x*-ray treatment they had received. The anterior longitudinal ligament remained normal in each case and was not, in his opinion, responsible for the "bamboo spine." Each vertebral body was connected to the adjacent one by a bridge of smooth bone, and the intervertebral disk was swollen; around its periphery there was a heavy deposition of calcium salts. The calcified tissue eventually became ossified, and in his view this formed the typical "bamboo spine." Cartilaginous proliferation, abnormal calcification, and excessive new bone formation were found in other joints. There was no synovial proliferation, and this seemed to be a further indication that spondylitis and rheumatoid arthritis were distinct disease processes.

"Tension Syndrome"

Dr. R. M. MASON and Dr. W. S. C. COPEMAN (London) reviewed current hypotheses on the pathogenesis of non-articular rheumatism. They then described the "tension syndrome," a condition which had previously been confused with "panniculitis"; it might ultimately lead to localized fibro-fatty herniation. Attempts had been made to exacerbate the symptoms by deliberately inducing water retention with deoxycortone acetate. This has proved ineffective, but cortisone given in a single large dose both reproduced and exacerbated the pains of three patients suffering from this syndrome. Intravenous hypertonic saline had a rapid though temporary effect in relieving the pain in two cases. The physiology of intracellular and extracellular fluid retention was reviewed, and this was correlated with the experimental results reported.

Dr. DORIS BAKER (London) also reviewed this clinical syndrome and discussed its pathology, as determined by skin biopsy, its differential diagnosis, and its treatment. It was postulated that it might be an endocrine dysfunction associated with an excess of collagen in the dermis, with sodium retention.

Brucellar Rheumatism

Professor A. PEDRO-PONS (Spain), Dr. A. ROBECHI (Italy), and Dr. R. CIRERA VOLTA (Spain) gave extensive reviews of the bony and arthritic manifestations of brucellosis: in addition an assessment of some of the newer antibiotics in these conditions was given. "Aureomycin," chloramphenicol, "terramycin," and streptomycin all suppressed the disease, but relapses were frequent, and active immunization with intravenous vaccines was recommended.

Ultrasonic Therapy

Dr. A. BÖNI (Switzerland) and Professor K. MATTHES (Germany) discussed contemporary views on ultrasonic therapy, which had been tried empirically in most rheumatic conditions. It was considered that the effects were non-specific, the useful sedative action being probably due to deep, localized heat.

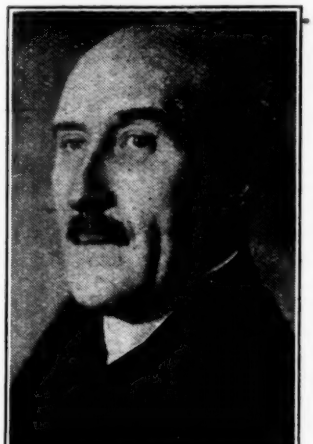
Endocrine Aspects of Rheumatic Diseases

Dr. C. H. SLOCUMB (U.S.A.) reported a series of 100 cases of rheumatoid arthritis treated with cortisone. Dr. C. HAMBURGER (Denmark) described experiments designed to relate the dose of adrenocorticotrophic hormone (A.C.T.H.) to the metabolic response as measured by the increase in urinary corticoids and 17-ketosteroids and the fall in eosinophils. In all but one case the metabolic response

was related to the frequency of injections rather than to the total dosage, and the need for a satisfactory "retard" A.C.T.H. was stressed. Dr. GUNNAR EDSTRÖM (Sweden) reported experiments in man with pituitary transplants from calves and pigs. When the transplant survived it caused all the expected metabolic effects. In addition, the implants had had marked but temporary therapeutic effects in cases of rheumatoid arthritis and disseminated lupus erythematosus, but no claims were advanced that this could become practical therapy. Dr. J. H. KELLGREN (Manchester) described a comprehensive study of the articular and other limb changes in acromegaly. Biopsy studies suggested an increase in soft-tissue collagen. Dr. G. D. KERSLEY (Bath) compared the clinical and metabolic effects of hypoglycaemia and A.C.T.H. in the treatment of rheumatoid arthritis, and Dr. L. MANDEL (Bath) reported a series of cases treated with intra-articular cortisone.

CAPTAIN CROOKSHANK

The new Minister of Health and Leader of the House of Commons, Captain H. F. C. Crookshank, was born in 1893. He was educated at Eton, where he was a King's scholar, and at Magdalen College, Oxford. After serving in the 1914–18 war with the Hampshire Regiment and the Grenadier Guards in France and Salonika—he was awarded the Order of the White Eagle and the Serbian Gold Medal for Valour—he joined the Diplomatic Service in 1919 and was attached successively to the Foreign Office and the British Embassies in Constantinople and Washington. In 1924, on his election to Parliament as Conservative Member for Gainsborough, he resigned from the Diplomatic Service.



Elliott and Fry, London.

Captain Crookshank is above all else a Parliamentarian. His profound grasp of procedure in the House of Commons and his cool adroitness when in difficulty have been displayed there since his first entry into the House. Hitherto he has not specialized in health matters. He was Financial Secretary to the Treasury from 1939 to 1943 and has long spoken with authority on this subject. There, as in the other posts he held, as Under-Secretary for the Home Office (1934–5), Secretary for Mines (1935–9), and Postmaster-General (1943–5), he was administratively efficient in his Department and watchful against extravagance. He has shown the same watchfulness in the House of Commons, where his speeches have been noteworthy for clarity, pointedness, and imperturbable temper and a quick wit, and he enters his new office with the respect of all parties.

He is a Knight of the Order of St. John of Jerusalem.

Most of the 112 holders of Fellowships awarded by the United Nations and its specialized agencies, who are now studying in the United Kingdom, attended a United Nations Day gathering at the British Council's London office on October 24, where they met people concerned with their training in this country. They come from 33 countries, and the ambassadors and high commissioners of these countries were also invited. Dr. Gilbert Murray received the guests. The Fellowship holders are studying social welfare, various aspects of economic development and public administration, and medicine. Since 1947, 695 Fellows have been received in the United Kingdom.

Reports of Societies

STATUS OF GENERAL PRACTICE

At an ordinary meeting of the Liverpool Medical Institution, held on October 18, with the president, Dr. Robert Coope, in the chair, Dr. K. Cobban and Dr. S. P. C. O'Reagain opened a discussion on "Problems of General Practice."

Dr. COBBAN said that the family doctor held a responsible and proud position. He was much more than a medical adviser; he was a family friend and counsellor to whom confidence and trust were given. Unfortunately his training gave him no knowledge of the problems of general practice, and did not help him over his first difficulty—to obtain the confidence of his patients. Dr. Cobban then gave examples taken from an actual surgery list to illustrate the problems arising to-day.

Divorce from the Hospitals

The relationship with hospitals was raised. The offhand treatment of general practitioners was exemplified by the delays in sending, or even the complete absence of, reports. The unnecessary appropriation of patients by hospitals was deplored, and it was urged that not rivalry but collaboration should be the aim. It was contended that part, at any rate, of the present diminution of the clinical field of general practice was unnecessary, and that there was urgent need for direct access to diagnostic facilities and to hospital beds. It was only too easy to refer anything at all difficult to hospital, with resulting loss of clinical acumen, of sense of responsibility, and eventually of professional interest and ability to think.

In midwifery there had been loss both of skill and of responsibility which ought to be borne by the family doctor. The need for general-practitioner maternity homes was noted, these being considered better than hospitalization for the normal case. The ultimate responsibility for the patients in these or their own homes should rest with the general practitioners, and it was an unfair burden to lay on the midwives.

The recommendations of the B.M.A. committee on the association of general practitioners with hospital work were briefly discussed. Their importance lay in the attempt to facilitate collaboration with the hospitals, to bridge the gap between the general practitioner and the specialist, and to give general practitioners opportunity to cultivate some special interest. This last point had always been encouraged in theory, and had been carried out successfully in the past mainly in country districts with cottage hospitals. Under present conditions it was discouraged and the openings were appropriated by specialists. The new proposals might revive and improve the old system, but personal experience suggested that hospital appointments to be of value to general practitioners must carry responsibility and fulfil a necessary function in the hospitals concerned.

But it was impossible to arrest loss of skill and responsibility by those methods so long as lack of time prevented their use. The need for haste enforced reference to hospital, and prevented study and adequate consideration of patients. General practitioners were in danger of losing their position not only as medical advisers but also as family counsellors and friends.

The Financial Side of Practice

Dr. S. P. C. O'REAGAIN said that an organization was required to deal with patients expeditiously. This meant that the general practitioner must be a business man and direct his staff and watch expenditure efficiently; he was not always experienced in business and must be prepared for stress, financial and otherwise. The doctor–patient relationship was marred by some mercenary considerations

one being that each patient must be regarded as a potential litigant. This might mean frequent radiographs in cases of minor injuries, particularly those sustained at work, thus increasing the work of the general practitioner and the casualty department. The appearance of practising one's art must not be neglected; this might lead to time-consuming over-examination of healthy people and wasteful prescribing. Otherwise the doctor–patient relationship was satisfactory and the general practitioner was still regarded as the family doctor.

Clinical Problems

Tuberculosis in general practice was discussed. It was a matter for regret that the general practitioner could not find time to accompany his patients to tuberculosis and other clinics; because of this the doctor could only follow directions of treatment from the specialist. A plea was made that large film chest radiographs might be made available as a direct general-practitioner service.

Certification in general practice was described and discussed. It was accepted as a necessary part of the work and in the patient's interest, but many certificates of a non-medical nature could be relegated for signature to people other than the general practitioner, whose aid was often sought first as being the easiest to obtain. Finally, Dr. O'Reagain declared that, though he had spoken at length on the problems of general practice, he could speak much longer on the joy and satisfaction to be derived from the practice of this branch of medicine.

Pathological Services for G.P.s

In the subsequent discussion Dr. D. A. STANLEY said that his experience of providing pathological services to general practitioners in another area before coming to Liverpool was that those general practitioners who used the facilities before the appointed day under the old private practice arrangements continued to use them with very little increase; those who had never felt the need for pathological facilities under the old system continued not to feel the need; and that as new general practitioners set up in practice in the district they usually requested facilities and used them. There was no difficulty in providing such services—all that was needed was a little extra technical help and a little more accommodation. The finances of management committees had made even this not available.

Dr. J. D. CRAIG agreed with Dr. Cobban on the great value of having a few beds, on cottage hospital lines, available to the general practitioner; they would ensure contact between consultants and practitioners to their mutual advantage. He felt that house-officers should be instructed to accept the word of practitioners on the admission of emergencies so that early diagnosis would be encouraged. If the admission was unnecessary, discharge could be effected next day by a senior officer.

Value of a Chance to become a Consultant

Dr. P. J. GIBBONS said it was scarcely in dispute that the status of the general practitioner had declined since July 5, 1948, and, though on all sides that was deplored, very little had been done to remedy it. There was one simple and obvious remedy, which would immediately go a long way both to restore the status of the general practitioner and to reunite the profession, which had been sadly divided by the Act of 1948. This was a return to the system which had existed before 1948, by which a man in general practice who wished to become a consultant had a road through general practice on to consultant status. It had to be admitted the road was a hard one, but it was there for those who wished to travel it. That road had been completely closed since the appointed day. To reopen the road would present some difficulties, but none of them were insurmountable, given good will. The simplest way would be to create clinical assistantships for younger practitioners, and to create enough of them so that a reasonable choice could be exercised by those in general practice. Hospital authorities and medical boards should be empowered to appoint a man from general practice to full consultant status when his work and merits deserved it.

A Consultant's View

Dr. E. T. BAKER-BATES, speaking as a consultant physician, said he believed that a true sense of values in medicine was never more needed and never harder to maintain than at present. In teaching hospitals there was a tendency to spend too much time studying highly selected material, and not to see or teach the day-to-day problems of sickness. The commonest disorders of daily practice were seldom seen in hospital. The student was rarely taught to assess subjective symptoms, to unearth anxieties, and to handle the sick as seen in general practice. Modern hospital practice and teaching in many instances did not really equip the future general practitioner, and many consultants were ignorant of the problems which prevailed in general practice.

Facilities for investigation should be available for the general practitioner; this would lessen the demand on the consultant services. But at the moment all the resources of the hospitals were strained to the limit and would break down if additional demands were made. It would be difficult for many practitioners, especially those in large working-class areas, to take proper advantage of such additional facilities, as the demands of many patients on the time of the doctor made sound clinical medicine almost impossible. Clinical assessment of the individual patient was essential before any investigation could be undertaken; it was a skilled and time-consuming procedure, and there was no short-cut to diagnosis. Many of the profession were under the illusion that a correct diagnosis could always be made by a series of investigations. The opinion was widely held that the onset of a lesion was associated with radiological signs. Many findings on investigation were not responsible for symptoms, and good judgment was required to assess the significance of abnormalities found on investigation. Special tests could only be interpreted in their clinical context, and the potential source of error was great. Much unnecessary alarm and treatment would be the consequence of indiscreet interpretation of investigations.

Dr. Baker-Bates considered that beds should be available in hospitals where the general practitioner could treat patients who could not be adequately looked after at home and did not need skilled investigation and specialist supervision. Criticism had been made of the "follow-up clinic." It was desirable for the consultant to keep under observation cases where the diagnosis was in doubt, certain cases of academic and teaching interest, and those patients being treated with powerful and potentially toxic drugs such as cortisone and thiouracil.

Dr. C. M. OGILVIE thought that closer liaison was needed between the general practitioner and the out-patient staff of hospitals in arranging for the supervision of further treatment of patients after discharge from hospital.

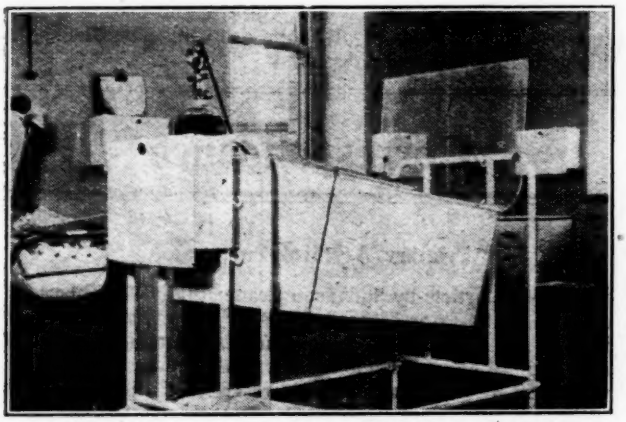
The Philosophy of General Practice

Dr. P. STRELITZ recalled that it had been said medicine was lovely to know but difficult to practise. He felt that the more medicine one knew, the more difficult it was to practise outside a hospital. There were no problems of general practice as such—the problems were those inherent in the personality, ambition, and ability of the doctor, and both Dr. Cobban and Dr. O'Reagain had obviously solved their own problems. The only satisfaction in medicine was to practise in a limited field, and for that reason the practitioner should confine himself to, and specialize in, general practice, which should be a leisurely vocation. He should specialize in patience and kindness and getting to know his patients, and be able to bring the personal touch to bear and to gain his patients' full confidence. In that manner no major problems would arise.

"NO TOUCH" AND THE NEONATE

The Paediatric Section of the Royal Society of Medicine opened the new session on October 25 with a combined meeting with the Section of Obstetrics and Gynaecology to discuss "The Care of the Newborn Baby." Professor A. G. WATKINS, the president of the section, opened the discussion by describing the methods adopted by Dr. J. Jacobs and himself in the routine care of babies at St. David's Hospital, Cardiff.

Just under 2,000 births a year out of a total of about 4,500 in Cardiff took place in the maternity department of this hospital, which included a unit for premature babies and a breast-milk bank. Newborn babies were under the day-to-day care of a consultant paediatrician and a paediatric sister. Cots of the Sorrento type were used, modified by the addition of a hinged "perspex" lid (see Fig.) and a "perspex" shield to prevent the baby coming into contact with hot bottles. Nursery temperature was maintained at 70° F.



Modified Sorrento cot, showing hinged "perspex" lid.

(21.1° C.), with the cot temperature increased by bottles. The "perspex" lid allowed the cot to be used as an oxygen bed if necessary, while still providing easy access and a clear view of the baby.

The First Few Days

On receiving a baby from the labour ward, after immediate resuscitation he was placed without bathing and without clothing (except a napkin) in his cot. The cord was cut short—about ¾ in. (1.8 cm.)—three or four hours after birth, and re-ligated, collodion being applied to the stump. No cord dressing, binder, or powder was used. The baby normally received its first bath six to eight days after birth, after the cord had separated. No prophylactic eye treatment was given, with a consequent reduction in the incidence of "sticky eyes."

Delayed feeding, at first successfully used with premature babies, had been extended to the normal full-term infant, who was not fed until there was evidence of hunger—a characteristic cry, restless movements, and sucking of the fist. The average period before these signs appeared was 35 hours, but might be longer. The first feed consisted of saline, to test the continuity of the alimentary tract and the cough reflex, and after this the baby, clad in a single light garment, was taken out of the nursery and transferred to a cot at the foot of the mother's bed, where he usually remained until mother and child returned home. The first bath was given at the bedside of the mother, who thereafter bathed the baby herself. There had been no trouble due to engorged breasts associated with late feeding, and this was thought to be largely due to the work of the lactation clinic, which was supervised by the paediatric sister and also provided later the majority of donors for the milk bank.

With this "no touch" technique in the early period, there had been steady reduction in sepsis; deaths attributable to infection had been, in 1948 10, in 1949 6, and in 1950 3.

Dr. J. JACOBS described the arrangements in more detail, showing the fall in neonatal mortality during the last three years as compared with previous years. He also showed figures demonstrating an improvement in the number of babies discharged fully breast-fed (from 89% in 1949 to 93% in 1951) and charts showing details of the breast-milk bank; about 250 pints of milk were collected weekly, pooled and pasteurized, and mainly used in the hospital, especially in the premature unit. It was essential that the donor should be healthy and be successfully feeding her own baby. Dr. Jacobs also showed a model of the modified Sorrento cot, and showed, too, detailed charts demonstrating the marked reduction in the number of "sticky eyes" needing treatment since prophylactic eye drops had been discontinued.

Control of Pemphigus

Mr. J. B. BLAIKLEY, for the obstetricians, gave a detailed account of measures adopted to prevent the spread of staphylococcal neonatal infection by bacteriological control. This had been undertaken at Guy's Hospital, following outbreaks of neonatal pemphigus. Frequent swabs were taken from hands, nose, and throat of all patients and other persons entering or working in the department, and any staphylococci found were classified according to the grading evolved by Dr. F. A. Knott. This grading, based on biochemical activity, seemed to have some advantages over phage-typing, especially in that the results came through more quickly. Almost all severe infections causing epidemics or leading to lesions needing treatment were found to be due to staphylococci of Knott's grades A and B; and patients found to be carrying such organisms were isolated, while if these were present in swabs from staff or other personnel the carrier was excluded until clear. Mr. Blaikley agreed that bathing of babies should be kept to a minimum, and placed great emphasis on a strictly observed hand-washing technique.

Discussion on Nursery Technique

Dr. A. WHITE FRANKLIN made the point that the gratifying improvement in neonatal mortality and morbidity given by Professor Watkins was perhaps not so much due to new departures in technique as to the fact that a new and active interest had been taken in the welfare of the babies, all of whom were receiving skilled and constant observation.

Professor G. I. STRACHAN congratulated Professor Watkins on his sedulous care of the babies in Cardiff, and noted how at last, and during his time, the wheel had come full circle: babies had again returned from the nursery to be kept at the mothers' bedsides.

Dr. MARY CROSSE wondered how they persuaded the nursing staff not to bath the babies. Nurses loved doing this, and if they did less of it they would have more time to wash their own hands.

Dr. BERYL CORNER emphasized the importance of the babies being free to move their limbs in the early period, especially the arms. Arm movement seemed to be an important help in establishing normal respiration.

Dr. H. WALLER pointed out that delay in putting the baby to the breast for 35 hours did not really constitute "delayed feeding," because the baby received little or no milk from the breast until after this period.

Dr. HELEN MACKAY and several others raised the question of masks and whether they were really necessary.

Conclusions

The conclusion that seemed to emerge from the meeting was that, while the new techniques described were an improvement, and certainly saved much time, the important recent advance in the care of the newborn consisted in their careful and detailed observation, leading to the early detection of abnormalities; and also in the increased attention to the smaller details of necessary nursing care which resulted from the appointment of a sister with special charge of the obstetric nursery. The baby, as Professor Watkins and Mr. Blaikley agreed, should be regarded as just as much entitled to detailed examination and care as any other patient in the hospital.

ALCOHOL

A meeting of the Nutrition Panel of the Society of Chemical Industry on October 24 was addressed by Dr. R. P. COOK, Lecturer in Biochemistry at University College, Dundee, on the subject of the physiology and psychology of alcohol.

Dr. Cook said that man appeared to feel the need for mental and physical stimulus. In some persons this found satisfaction in intellectual and bodily activity; others resorted to drugs. The call of civilization for stimulants or stupefiers was reflected in the inland revenue figures for the financial year 1949–50, when the duty on beer yielded £263m. and that on spirits £59m., not to mention £602m. from tobacco.

Absorption and Metabolism

Alcohol was produced by microbial action. It was readily absorbed—about one-fifth from the stomach, one-tenth from the upper part of the small intestine, one-half from the middle part, and one-fifth from the lower. The speed of absorption seemed to depend upon the form in which the alcohol was taken, particularly its dilution, and also upon the time since the last meal. Mellanby found that a fixed dose of alcohol was more rapidly absorbed when given at 20% concentration than at 5%. Food in the stomach diluted the alcohol, but the type of food was important; bread and milk delayed absorption, but if the fat was removed from the milk absorption was quite rapid. Alcohol excited the secretion of gastric juice, but concentrated alcohol produced gastric irritation, and might cause eventual inhibition of gastric secretion lasting for some days. Alcohol was oxidized at a constant rate, mostly in the liver. There was no evidence that it could be used in muscular exercise, and it seemed unlikely that enzyme adaptation took any part in the process. The amount of alcohol lost in sweat was said to be small, but might be an important factor in manual labourers.

That alcohol was not the sole cause of cirrhosis of the liver was proved by the occurrence of this condition among abstainers, but in chronic alcoholism the first stage was fatty infiltration of the liver, and, following this, cirrhosis—or more properly fibrosis. Lipotropic factors had been found by Best and his colleagues to prevent this occurring. Why alcohol should attack the higher centres was unknown, but it was possible that the "enzyme geography" of those centres was more complex and therefore more easily upset.

"Under the Influence"

When persons were charged with being under the influence of drink while driving a car differential diagnosis was important. Similar symptoms might be due to an overdose of insulin in a diabetic or to uraemia. Behaviour tests were apparently the most important evidence in court, but it should be borne in mind that even for some normal persons not under the influence of drink the tests might be more severe than for others. Methods had been elaborated for measuring the alcohol in the blood, but urine was easier to collect, and there was a relation between the concentration in urine and in blood. In the United States a "drunkometer" was used to measure the alcohol in the breath. It depended on the amount of breath required to decolorize a known volume of potassium permanganate.

Why did certain persons develop a taste for alcohol? Psychiatrists had various explanations. One of them had pointed out that in young persons nourishment in the emotional sense was connected with sweet things, and eventually alcohol might be substituted for sweets; in support of this it was noted that the type of alcoholic beverage often favoured by the young was a sweet drink. In adolescence alcohol might be regarded as symbolic of manhood, and taste might develop for a bitter drink. It was easy to understand how a conditioned reflex or what almost might be called an obsessional neurosis was set up. Alcohol was often used as a means of escape from unfavourable surroundings. The latest theory was that alcoholism was associated with adrenal insufficiency, and it was claimed that good effects in its treatment were obtained from cortical extracts. Finally Dr. Cook discussed remedies or palliatives. A saline drink was said to prevent a "hangover." One remedy for alcoholism which had been put forward was tetraethylthiuram disulphide ("antabuse").

In the discussion which followed Dr. Cook's address a divisional police surgeon said that previous head injury was, in his experience, one of the most important factors making for abnormal behaviour; if the injury had been sufficient to produce unconsciousness it predisposed these persons to a greater effect from drink. The same speaker also suggested that in testing for drunkenness the eye test was of chief importance, though often disregarded. If a man could not see a number-plate at 25 yards (22.8 m.), equivalent to a vision of 6/15 on the chart, he ought not to be driving a car, whether he was under the influence of drink or not.

Mr. A. L. BACHARACH suggested that other constituents of drink besides the alcohol might have some pharmacological action, though commonly ignored. There was ignorance on many aspects of the alcohol problem, and in view of the immense consumption of alcoholic drink by normal people this ignorance was to be deplored.

Correspondence

"Primary Atypical Pneumonia"

SIR,—The article by Squadron Leader Philip W. Robertson and Dr. K. D. Forgan Morle (October 27, p. 994) is of great interest and also calls for comment. All attempts to throw light upon the syndrome of atypical pneumonia will be welcomed by those genuinely interested in the interpretation of respiratory tract syndromes. But the authors of this new attempt are at such pains to ridicule the virus theory of aetiology that they appear to have overlooked certain facts. They ask why a virus should select isolated dependent lung segments for its attack. But such selection occurs normally in animals inoculated experimentally with material containing a virus capable of attacking the lung. In fact, aspiration of fluid placed on the nostril after anaesthetization is the normal technique of inoculation practised with either influenza or any of the animal viruses capable of attacking the respiratory tract. Inoculation of mice intranasally with influenza virus fails to induce lung lesions unless an anaesthetic is used. Moreover, lung lesions do not develop if the virus-containing fluid is previously mixed with immune serum. No one therefore doubts that the experimental "aspiration" lesions are caused by the virus infection. Exceptionally, as with certain highly virulent influenza strains in the ferret, lung lesions develop even without an anaesthetic, and the use of anatomized virus suspensions will avoid the need for an anaesthetic in mice.

Are these facts entirely without significance in human pathology? The need to bring the virus into mechanical contact with the bronchus and alveolus holds for the experimental animal. Is it not possible, in the case of some viruses normally inhabiting the human upper respiratory tract, that the lung is not attacked unless the virus is induced mechanically to descend down the trachea? Nevertheless in the case of Q fever and of infection by the ornithosis-psittacosis group of viruses the exact mechanism for the production of lung lesions is not obvious. Surely Drs. Robertson and Morle do not deny that the lung lesions are related to infection by the organisms causing these diseases. Their own evidence in relation to common respiratory infections is impressively in favour of the mechanical results of aspiration. We should be as unwise to accept all cases of the atypical pneumonia syndrome as being "non-specific" in aetiology as to assume the existence of a ubiquitous pneumotropic virus.—I am, etc.,

Sheffield. C. H. STUART-HARRIS.

Plain Words

SIR,—It is right, no doubt, in the interest of lucidity to chide, decry, impeach, censure, execrate, or even denounce official phraseology when the construction is incomprehensible; and it is justifiable up to a point to prefer comparative simplicity when nothing is in consequence sacrificed. But I think the present-day campaign is overdone.

For I see in it some support of a modern tendency to save trouble and make do. We have utility furniture, utility clothing, utility alimentation, so why not utility language? It is so convenient to have one word to serve all purposes. A superficial observation of everyday conversation invites the conclusion that only two adjectives are in current employment. "Marvellous" to describe all circumstances and qualities, from the mildly interesting to the most extraordinary: "appalling," for all occurrences from a trivial inconvenience to an extremely terrifying experience. And, since official language in an anxiety to be comprehensive and convey a precise shade of meaning inevitably includes a number of more or less unusual words, critics encourage the development of the verbal miser who hoards up the riches of our vocabulary between the boards of dictionaries and subsists on battered or counterfeit phraseology. The courteous tradesman who "regrets that he cannot comply with your request" encounters the sneer that his meaning could be just as well conveyed by "sorry, no can do." He may even be reminded that by his more laconic version he saves 1.63 second—doubtless of great importance in these hustling times. It may be an old-fashioned prejudice to express a preference for the more elaborate version.

Dr. Bernard Isaacs (October 27, p. 1032) thinks that our profession deserves special condemnation; partly for its specialized jargon, principally, I gather, for its unnecessary circumlocution. Criticism and ridicule of anyone's composition and use of words (even mine) are not particularly difficult. For, although to speak and to write fairly good English is a not over-laborious occupation, to speak and write what the pundits admit as impeccable is a nightmare of study. To take into consideration what is etymologically accurate, what is legitimately acceptable through custom, what is completely unambiguous, leads to a sort of neurosis when one is afraid ever to open one's mouth or put pen to paper. And for the most part I think the majority of our perpetrations are, if open to criticism, comparatively venial. Dr. Isaacs confesses to a favourite list of overworked and ill-treated words and phrases—a weakness to which we could all plead guilty—and I do not find him very convincing.

He dislikes the description of "pneumonia being superimposed on an underlying bronchiectasis." Well, what would he substitute? He selects for condemnation "association" by appending two examples: "tuberculosis in association with pregnancy" and "a cold in the head in association with an ingrowing toe-nail" and asks why not just plain "and"? With all respect, "and" would be as incorrect in the first example as "association" in the second.

I would hesitate to join a Society for the Employment of Correct English, but if Dr. Isaacs will institute a Society for the Prevention of Cruelty to English I shall enthusiastically apply for membership. For I think it would be better so far as our profession is concerned to cast a lenient eye upon the grammatical misdeeds for which we are stigmatized and apply ourselves to proscribing the slovenly inelegant professional slang only too likely to be perpetuated by the present attack upon officialese.

Eliminate such horrors as flu, gynae, primip, abdo, pul. tub., auric. fib., chronic belly, a cardiac, teeth O.K., bismuth-mealed, she had a Caesar, do a P.R. That achieved, we might proceed more ambitiously in the direction of style, gently pointing out that there are other adjectives than "marked," other verbs besides "to get." *Obsta principiis*, for the habit is acquired early in a student's training. If one asks for the description of a lesion, in the vast majority of cases the construction proceeds on the lines—"You get." I may resent the imputation when he is referring to hobnails in the liver, or reject the absurdity if he assures me that "You get haemorrhage from the vagina." That perhaps is as far as I, a pusillanimous creature, would be prepared to go to avoid being at first distrusted, and end by being thoroughly detested.—I am, etc.,

London, W.1. ADOLPHE ABRAHAMS.

SIR,—We must reluctantly admit that Dr. Bernard Isaacs (October 27, p. 1032) is right. Medical jargon has increased, is increasing, and ought to be diminished. The remedy lies in the hands of teachers of clinical medicine. If they studied the style in which their students' reports were written, as well as the facts they contained, circumlocution should soon be merely a memory.

In *Plain Words*, his first booklet on the writing of English, Sir Ernest Gowers comments on the growth of Puddery, a word Ivor Brown coined to describe "saying what one has to say in as complicated a way as possible."

"Puddery seems to be regrettably increasing in medicine. In my lifetime I have seen the *mad-doctor* pass through the chrysalis of *alienist* into the butterfly of *psychiatrist*. This is perfectly excusable, but why have *walking cases* suddenly become *ambulants*? Some seventy years ago a promising young neurologist made a discovery that necessitated the addition of a new word to the English vocabulary He insisted that this should be *knee-jerk*, and *knee-jerk* it has remained, in spite of the efforts of *patellar reflex* to dislodge it. He was my father; so perhaps I have inherited a prejudice in favour of home-made words."

If anyone is tempted to begin a sentence with the words, "in the case of," he should read the amusing lecture on jargon delivered by the late Sir Arthur Quiller-Couch. He quoted a report from the clerk to a board of guardians which read, "In the case of John Jenkins deceased, the coffin provided was of the usual character." "Q" pointed out that "actually John Jenkins never had more than one case, and that was his coffin." Fowler remarks that this phrase can often be omitted without altering the meaning of a sentence, and adds that its use is merely "the lazy impulse to get the beginning of the sentence down." Again, are we always as careful as we should be in the choice of a word? John Jenkins's coffin could have had no character unless it possessed distinctive marking. Then it would not have been "usual."

Dr. Jowett is said to have given a servant a character in which he remarked that he was "generally good." The man behaved badly in his next place, and Jowett received an angry letter asking what he meant by describing this man as "generally good." To which he replied that if the writer would consult a dictionary he would find that the definition of "generally" was "not particularly."

There is an innovation which must be checked before it is too late. Sir Alan Herbert has already protested against the unwarranted manufacture of new verbs. He writes: "There seems to be a notion that any British or American subject is entitled to take any noun or adjective, add *ize* to it, and say: 'I have made a new verb. What a good boy am I.' This notion must be 'finalized.'"

You, Sir, are not free from blame. You have allowed a contributor to write on p. 1015 of the current number an appalling sentence beginning, "Here Latin and Greek are anatomized . . ."

Surely this is an even more deplorable example of ugly English than the use of "bed-dage" which you once wittily criticized. If this verbal prostitution is allowed to continue unchecked, we shall wake up one day, as you suggested, to find our officials referring to "hot-water-bottle-age."

There is a real danger that many more manufactured verbs will appear. The English language is always growing, and it is good that it is. But no writer with an ear for the music of language should invent and use ugly words.—I am, etc.,

London, N.W.1. IVO GEIKIE-COBB.

*** The word "anatomized" is no new invention. Current in the sixteenth century, it has been commonly used ever since in ordinary educated speech and writing—for instance, by Shakespeare, Burton, Cibber, Burke, and Browning.—ED., *B.M.J.*

What Makes Hair Grow?

SIR,—I should like to congratulate you on the concise reply to this difficult question ("Any Questions?" October 13, p. 919), but do not agree with the author's explanation

of the beaded hairs in monilethrix. The theory expounded was originally suggested by G. Behrend (*Berl. Klin. Wschr.*, 1885, **22**, 270) and demonstrated by G. Ciarocchi in 1900 (*Ann. Derm. Syph., Paris*, 4 ser., t.1, 1028). These workers believed that the thick nodal portion was produced during the night in the absence of nervous tension, while the constricted part was grown during waking hours.

In a case I recently studied (*Brit. J. Derm. Syph.*, 1950, **62**, 35) this attractive theory could not be supported by experiment. In the first place, by shaving a small area of the scalp and examining the growth after two weeks the hair shafts did not reveal 14 nodal complexes. Furthermore, by keeping the patient awake for 36 hours there was no interruption of the regular beading to be found.

One important factor in stimulating growth of hair is general exposure to sunlight, though this is not apparently due to vitamin-D synthesis alone.

The factors governing the length of hair growth are still unknown. In this connexion it is of interest to note that while it is possible to regrow scalp hair in some early cases of premature alopecia in males by oestrogen therapy, these hairs seldom exceed 1 in. (2.5 cm.) in length.—I am, etc.,

London, W.1. IAN MARTIN-SCOTT.

Hairiness in the Young

SIR,—You have been kind enough in the past to permit me to seek help from your readers in some aspects of endocrinological research by accepting a brief letter of a similar character to the present. At the moment I am investigating hairiness in infants and children of both sexes up to the age of 16, and I should be pleased and appreciative if any such cases, not already being specially investigated elsewhere, were sent to see me at the Princess Louise Children's Hospital, St. Quintin Avenue (Ladbroke Grove), on the first and third Thursday afternoons of every month. The appointment system is in operation, and a telephone call to LAD. 0133 would result in a definite time being given.—I am, etc.,

London, W.1. S. L. SIMPSON.

Educating the Public about Cancer

SIR,—May I draw attention to the following statements by a leading member of the profession in an article contributed to the current public monthly *Family Doctor*? "Each year about 20,000 patients are cured of cancer in England and Wales alone. In the case of breast cancer the best results show that 75 out of every 100 early cases are cured, but in the late stages only 19 out of 100 can be cured."

Most medical men from long experience know that they cannot say one case of cancer in a hundred has been cured by them, and the members of the public who have trusted their doctors have had a similar experience. It is very doubtful if a doctor has any right to claim that he has cured one case of cancer. In my experience prompt surgical removal of the earliest primary cancer has failed to cure; death due to secondary cancer has occurred within a variable time extending from a few months to several years. It is no excuse to call it a "cure" if the patient survives three, five, or more years—some of the included years are but an existence. I have known patients with cancer untreated to live more years.

Our authority goes on to say, "You don't need a medical training to see that if these tumours are discovered when they are small and in one place, they can readily be cut out or destroyed by *x* rays or by radium." Who is competent to say it is "only in one place"? What did the surgeon, the late Percy Furnivall, say of the treatment of his early cancer? "I would not wish my worst enemy the prolonged hell I have been through with radium neuritis and myalgia for over six months." William Gosse made similar statements. But our authority says, "The chief reason for fear of cancer is ignorance." Neither the public nor the members of the profession fear cancer because of ignorance, if we exclude the ignorance we all possess of the disease and its true cure. They fear it because of such testimony as that of Percy Furnivall and of their own relatives and friends.

From my experience doctors—yes, and eminent surgeons with life-long experience of cancer—are probably less ready to come forward for investigation than members of the general public, for they know the serious limitations and possibilities of treatments. Our authority suggests that cancer develops in men and women who are perfectly healthy and unaware of the development. Yet from the earliest cases I have seen I have learnt that most intelligent patients can give a good idea when the development started; indeed, I would go further and say that even when they have reason to suspect it, and have gone to their doctors with their fears, unless the doctor has felt a lump, or got from his clinical and ancillary examinations some definite localizing sign, the patient can be subjected to every conceivable test and ultimately die before the site of the primary is found—indeed, it may not be possible even then to decide it. We do wrong to place the mentality of the patient on too low a level, or our own too high. We as doctors have good reason to show a little more humility in these matters.

"A breast cancer," says our authority, "does not grow in the night like a mushroom, so there is no sense in examining the breasts more than once in, say, three months." I might take this as an answer to the comment which I made in a former contribution (*Spectator*, October 28, 1949), when I repeated the advice of a leading surgeon that "women should carry out periodic inspections of their bodies for lumps, etc.—a sort of morning exercise."

Women have a nerve supply even in the breast, and the cancer manifests itself to their notice before the doctor could feel it, but, as with doctors, they find and fear it and hope against hope that the lesion will subside, as most of the causes of our fears do. Well might our authority say, "Indeed it helps the doctors to see you at your worst."

Little praise can be afforded to the inconsiderate consultant who says, "You know what's the matter, Missus: you've got cancer." Or he who chides the patient with an advanced lesion, suggesting to the distraught patient that if only she had come earlier he could have cured her. It does not follow that even if she had consulted him he would have discovered the lesion. I have known and recorded (*Medical Annual*, 1940, p. 507) the most eminent fail to make a digital examination of the rectum even when the patient has complained of continuous bleeding from the rectum and the feeling of a mass there. And in that case, even though a secondary carcinoma was reported as showing the characters indicating an alimentary tract primary, a useless hysterectomy was performed. It was because I insisted on a digital examination after the second operation that a large fungating carcinoma was found within an inch of the anal sphincter. The much-maligned disk has been removed when a digital examination of the rectum would have supplied the diagnosis. Because the specialist does not see the case until it is advanced he must not assume that the patient is stupid or, as is not infrequent, that the practitioner has failed to appreciate its significance.

This propaganda of fear has not even the merit of being original. It has been used in a similar manner in the U.S.A. for years. Twenty years ago it was prosecuted with the help of huge posters carrying the word "Cancer" throughout the hoardings of Paris, and for its support it had the pioneer work of France on radium, with its pre-eminent clinic. But in spite of all the propaganda, the early diagnosis, surgery, *x* rays, radium, and all other exhibitions, cancer continues to increase; where we have useful knowledge, as we have in tuberculosis, syphilis, etc., we get a progressive decline in the death rate.

Our authority began his article with the statement, "Each year in this country many thousands of patients are permanently cured of cancer."

There is evidence of similar propaganda of fear on tuberculosis in the August number of the same journal. By such propaganda we are creating dis-ease in our people. Sir John Charles has recorded in his report for 1949: "The amount of incapacity reported, which had increased sharply in the second half of 1948, remained above the amount reported before the start of the National Health Service, apart from the exceptional half of 1947."

The more and more varied specialists we have, the greater and more varied the appetite to appease, and the more need there is for propaganda which will frighten our people to seek medical examination and treatment, but may I suggest that such propaganda can only continue as long as we ignore the teaching of Ruskin's *Lamp of Truth*?

Find a cure for cancer and there will be no need for propaganda—our people will seek it at once. Until then let us adopt the quieter methods of our doctors who won the confidence and affection of their patients by a more humane approach.

A correspondent in the *Lancet* (September 8, p. 447) asks, "Is the freedom of the Press a myth?" I rather think it

is, since bona-fide criticism of some widespread medical practices is often refused, even by the medical press, which brings out the truth of a further statement of that correspondent when he says, "Far more serious than all the humbug is the suppression of important news because its publication might affect the advertisers in the paper." Could they be medical?—I am, etc.,

Birmingham, 15. JAMES F. BRAILSFORD.

Streptomycin in Gastro-enteritis

SIR,—I was interested to read of Dr. R. A. Shanks's experience with aureomycin and chloramphenicol in infantile diarrhoea (August 4, p. 272). The failure of chemotherapy to check the diarrhoea is certainly disappointing, but I would like to record the success I have had with oral streptomycin in the treatment of vomiting. Any infant with persistent vomiting is given one teaspoonful by mouth before each feed of the following mixture: 0.16 g. streptomycin in 1 oz. (28 ml.) of water. If the feeds are two-hourly then the streptomycin is given before alternate feeds. In the majority of cases the vomiting stops within 24 hours. During persistent vomiting anti-peristalsis of the stomach and gut occur, and coliform organisms must invade the upper gut and stomach in large numbers. I suggest that streptomycin suppresses or kills these organisms and corrects any irritation they may be causing. Others may have experienced the same results as I have, but for those who have not I think it is worth a trial. To stop the vomiting is half the battle in the treatment of gastro-enteritis. We rarely find it necessary to give intravenous fluid; intermittent subcutaneous injections of hyaluronidase are usually sufficient. However, I believe we see more "dietetic" gastro-enteritis in Malaya, and this is usually amenable to treatment.—I am, etc.,

The General Hospital, Penang. C. ELAINE FIELD.

Surgery of Parotid Tumours

SIR,—The exposure of the main trunk of the facial nerve in the presence of a normal parotid gland is not without its difficulties; these are increased in the presence of a parotid tumour, either benign or malignant. In our experience of exposing the facial nerve for the purpose of performing spino-facial anastomosis (Poppen, *Lahey Clin. Bull.*, 1939, **1**, 8) we have found it an advantage to strip from the mastoid process some of the anterior tendinous fibres of the sternomastoid muscle and nibble away the lower and anterior part of the process. This exposes the nerve where it emerges from the stylo-mastoid foramen without further dissection, and eliminates searching for it in the narrow space between the mastoid and the ascending ramus of the mandible. This removal of the mastoid process recalls the technique used by Fiolle and Delmas (*Surgical Exposure of the Deep-seated Blood Vessels*, 1921) in the exposure of the upper part of the carotido-jugular system.—We are, etc.,

CHARLES LANGMAID.
Cardiff. R. D. RICHARDS.

Bornholm Disease

SIR,—So far 17 cases have been diagnosed as Bornholm disease in this practice. The first was recorded on October 2. Only women or children have been affected. The distribution has been scattered, but where there are several children in a family they have usually all had it. The incubation period is difficult to estimate without exact knowledge of the initial exposure, but different members of a family, including children below school age, have been affected within 1–2 days of each other.

The usual course appears to be an abrupt onset of symptoms, with abdominal pain, headache, fever, and perhaps sore throat. In most cases the pain and headache have subsided within 12–36 hours with rest in bed, but the headache and sore throat are apt to persist for anything up to four days. Too short a period in bed has led to a relapse. A week appears to be necessary. The abdominal pain has been intermittent in every case, griping in character, and shifting about the upper abdomen. It has not affected the lower abdomen. Thoracic pain has not been noted. Abdominal pain may be very severe, is occasionally accompanied by vomiting, but not by diarrhoea. The tongue is clean. Abdominal tenderness is present where there is pain, and varies with its intensity. Headache has occurred in most cases, and has sometimes been the most distressing symptom. One patient who had a very severe headache became delirious for a few hours. The headache is sometimes mild and frontal, but is usually vertical or occipital. Sore throat, showing only mild faucial injection, has been noted in a few cases. Slight fever is usual, but temperatures of 103° F. (39.4° C.) were seen in one family. I should like to thank Dr. H. P. Lehmann for his permission to write of these cases.

As in the Oxford outbreak, a subsequent period of lassitude has been usual.—I am, etc.,

Wickham Market, Suffolk. R. J. CRUICKSHANK.

SIR,—The following cases, the symptoms of which suggest Bornholm disease with meningitic symptoms, occurred in a family of five living on a farm near Worcester.

The daughter, aged 5, while out in the fields in the afternoon of September 30, suddenly complained of severe pain in the region of the left loin, which doubled her up. The pain lasted for about half an hour and then disappeared. While in bed the same evening she had two similar attacks. Between one attack and the next she seemed to be quite well. The next day she was feverish and ill and had a severe headache with photophobia. She remained ill until about noon the following day, when she suddenly felt better; she was quite well next day. During the next eight days she had two similar relapses, with vomiting, fever, and pain on neck flexion.

On October 12 she was admitted to hospital with a temperature of 100° F. and slight neck rigidity, but Kernig's sign was negative. No other abnormality was found on physical examination. The C.S.F. was found to be turbid and under pressure: cells 610 per c.mm. (neutrophils 80%, lymphocytes 20%); chlorides 710 mg. %; glucose 54 mg. %; culture gave no growth. A white cell count and urine and chest *x*-ray examinations were normal. On the 13th she was much better, and the C.S.F. cell count had fallen to 430 per c.mm. During the next week her temperature gradually fell and she became quite well again.

The son, aged 2, suddenly woke at 1 a.m. on October 4, with severe pain along the right costal margin. His breathing was shallow, rapid, and painful; he was flushed and feverish. The pain lasted until 1 p.m. of the same day, when it suddenly disappeared. Within a few hours he seemed to be perfectly well again and has remained well.

The father, aged 46, first felt ill on October 7, when he developed a headache in the evening. Next day the headache was still present and his temperature was 101° F. (38.3° C.). He had a severe pain along the right costal margin and in the back which gradually disappeared. On the 12th and 13th he was very much better and was able to work on his farm. On the afternoon of the 13th he developed a headache again, and the pain in the chest recurred. Next day the headache was very severe; he had marked photophobia and sensitivity to sound, and he noticed difficulty in focusing on objects. There was no pain in the chest, but it recurred next day. His symptoms gradually abated, but he was unable to eat anything for five days. He has remained quite well since October 22.

The mother, aged 40, was well until October 8, when she suddenly had a severe pain along the right costal margin. Later in the day she became tender along both costal margins. The pain was aggravated by breathing; there were periods of up to four hours during which she was free from pain. The pain continued with intermissions during the next six days and she felt ill; on October 10 she had a slight sore throat. On October 14 she felt much worse and now had a very severe headache; the pain in the chest had, however, disappeared. The headache was continuous and severe and she occasionally vomited. On October 16 she also had severe low backache, and next day was admitted to hospital, looking unwell, but not acutely ill. Her throat was slightly injected; her temperature was 98.6° F. (37° C.). There was marked neck rigidity with pain on flexion, but Kernig's sign was negative. No muscle tenderness was found in the extensors of the spine, in the chest or the abdomen, and no other abnormality was found on physical examination. The C.S.F. was clear, pressure 70 mm. The pathological report was: cells 12 per c.mm.; protein 45 mg. % (globulin 18 mg. %); chlorides 750 mg. %; glucose 39 mg. %. Culture gave no growth. E.S.R. was 10 mm. (Westergren). White cell count and urine were normal.

During the next few days the severe continuous headache persisted and her temperature rose to 102.4° F. (39.1° C.), but by

October 20 the temperature was falling and her headache began to abate. She was discharged symptom-free on October 25.

The daughter, aged 8, was well until the morning of October 9, when she suddenly complained of severe epigastric pain. She became feverish and later in the day had a headache. Next morning she seemed to be quite well and remained so for the following two days. On the evening of October 12 she again became feverish, complained of a headache, and vomited several times. Next day she remained ill and was admitted to hospital with a temperature of 100° F. (37.8° C.). There was slight neck rigidity, but Kernig's sign was negative. No other abnormality was found on physical examination. The C.S.F. was found to be slightly turbid and under pressure: cells 163 per c.mm. (polymorphs 90%, lymphocytes 10%); protein 25 mg. % (globulin—no excess); glucose 43 mg. %; culture gave no growth. Next day she was much better and has remained well since then. The C.S.F. on October 23 contained 20 cells per c.mm.

It has not so far been possible to associate this outbreak with that occurring at present at Oxford; at least four of the cases there have had meningitic symptoms. Sporadic cases of benign lymphocytic meningitis have been occurring in this district throughout the summer and are continuing to appear. No direct contact has, however, been established with this family.

I wish to thank Dr. Geoffrey Aldridge and Dr. H. L. Milles for permission to publish these cases, and Dr. Patrick Kidd and Dr. R. J. Henderson for carrying out the pathological investigations.

—I am, etc.,

Worcester. T. D. LEWIS.

Tenosynovitis of the Wrist

SIR,—We have been interested to read the contribution by Mr. Maurice Ellis (September 29, p. 777) and the subsequent letters from Dr. James Cyriax and Mr. R. E. Shaw (October 20, p. 972). A recent study of ours (*British Journal of Industrial Medicine*, 1951, **8**, 150) involving clinical observations on some 544 cases may therefore be of some interest because of the conflicting views, both on the pathology and treatment of this condition. Although Mr. Ellis's post-mortem dissections suggest that there may be an extension of the tendon sheaths of the extensores carpi radialis high up into the forearm, this does not coincide with the accepted descriptions in standard textbooks on anatomy. Our biopsy findings failed to demonstrate more than the presence of some loose areolar tissue proximal to the upper limit of the carpal tendon sheaths, and we have been unable to confirm that the tendon sheaths extend higher into the forearm. Be this as it may in the cadaver, we are of the opinion that tenosynovitis affecting the abductor longus and the extensor brevis pollicis can occur as a separate clinical and pathological entity to the exclusion of the extensores carpi radialis, although in our experience the radial extensors of the wrist, together with the abductors and extensors of the thumb, are more frequently involved together (in our series 77% of all cases). His description of the mechanism of its production, while interesting, could apply equally to the muscles and tendons of the thumb, and, although other observers (Conn, 1931; Hammer, 1934; Cohen and Reid, 1935; Kulowski, 1936) have previously suggested that local anatomical factors have an important bearing on the aetiology of tenosynovitis, we consider that this view is untenable in that the lesion has hitherto erroneously been considered to be confined within the tendon sheath itself.

Although the pathology of tenosynovitis at the wrist is by no means clear, we believe that the essential lesion is a perimyotendinitis involving the musculo-tendinous junction higher up in the forearm and well above the generally accepted upper limit of the tendon sheaths themselves. The term "tenosynovitis" for this particular condition would therefore appear to be a misnomer which has been copied from textbook to textbook, and we would prefer the term "peritendinitis crepitans."

We agree that unaccustomed movement is the most frequent cause, accounting for 27% of our cases, other causes being resumption of work after absence (21%), and simple repetitive stereotyped movement (16.5%). Direct local trauma or an isolated muscle strain does not in our experience play a major part in its causation, although repetitive movement was an associated factor in 32% of all cases.

We would confirm that prolonged and rigid immobilization in plaster-of-Paris is contraindicated, being associated with a high relapse rate amounting to 36.7% in our series. We cannot confirm the statement by Dr. Cyriax that crepitus is never found in cases involving the carpal extent of the abductor longus and extensor brevis pollicis, if indeed this ever occurs, because in these cases the crepitus is always found proximal to the upper limit of the carpal tendon sheath itself on the back of the forearm. With the exception of De Quervain's stenosing tendo-vaginitis, which is an entirely different entity associated with obstruction within the transverse carpal ligament, we have never seen a case of simple tenosynovitis involving the thumb muscles take three months to clear up, and unless a mechanical obstruction is present within the tendon sheath itself we have never found it necessary to open the latter at operation in order to effect a cure. We hesitate to recommend deep massage in treatment and have indeed produced a recurrence with exacerbation of signs and symptoms by such methods. We are, however, in agreement with Dr. R. E. Shaw that restricted movement rather than rigid immobilization in plaster-of-Paris is the correct method of treatment, and our findings lead us to the conclusion that incomplete immobilization, either by strapping, or, better still, by the use of a detachable "perspex" splint in combination with daily full range movements, will, in the majority of cases, clear the condition up by the eighth to the tenth day irrespective of delay in commencing treatment. The employment of counter-irritants has never in our experience resulted in resolution of the condition in less than three weeks, but in more resistant cases we have found that short-wave therapy in combination with "perspex" splinting will accelerate resolution.

In situations other than the back of the wrist and forearm, to which the foregoing remarks apply, a true tenosynovitis may be precipitated by local trauma or other agencies, the lesion in this event occurring in the tendon sheath itself—for example, on the back of the hand.—We are, etc.,

A. R. THOMPSON.
L. W. PLEWES.
Luton, Beds. E. G. SHAW.

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College of General Practice

SIR,—To many fellows and councillors of the three Royal Colleges the proposal that there should be a College of General Practice may at first provoke a feeling of resentment; but as a member of council of one of these Colleges I want to give strong support to the ideals that have prompted the suggestion. We must uphold the traditions of general practice and make provision for higher training directed specifically to the requirements of this branch of medicine—as opposed to training acquired as a by-product of failure of our present higher diplomas. I doubt very much whether it will be wise to create another College, but every proposal made to promote and enhance the academic background of general practice will command the very warmest consideration.—I am, etc.,

London, W.1. REGINALD WATSON-JONES.

SIR,—The suggestion of Drs. F. M. Rose and J. H. Hunt (October 13, p. 908) that a College of General Practice should be formed promises to be the first genuine move towards reviving the morale of the G.P. But, if it is to succeed in raising our status (not so much with the public as with our colleagues—or should I say rivals?—in the hospital service), surely it will be necessary that it grant a diploma of similar standard to the M.R.C.P. or F.R.C.S.? It seems to me, for what the opinion of one individual may be worth, that such a college ought to grant a diploma of membership, perhaps by examination and thesis, after a qualifying period of not less than five years in general practice; and that such a diploma should, among other things, carry the right to inclusion on an executive council's obstetric list and to the employment of a trainee assistant.

After a further period of 10 years or so in active practice election to fellowship should occur—perhaps automatically: and this I submit should carry full consultant status, with the right to be called out in domiciliary consultation at public expense on the same terms as the hospital specialist. An arrangement such as this would bring three benefits: first, the G.P. would have a stimulus to improve himself and a goal at which to aim. Secondly, the senior man of experience and ability could replace some of his routine work by consulting practice, thus easing his own burden. And, thirdly, a valuable form of consultant opinion would become available for other G.P.s—a form of second opinion which is likely to become extremely rare when all specialists are exclusively trained in hospital and laboratory.

Of course the definition of the scope of general practice, which would have to precede the arrangement of an examination of this sort, would present difficulties. These, however, should be soluble provided that the examiners themselves are, or have been for the main part of their professional lives, in general practice.—I am, etc.,

Shrewsbury. JOHN C. RYLE.

SIR,—Competence in general practice can only be acquired by experience. It cannot be assessed by examination.

An early result of the proposed College of General Practice would be that the newly qualified man would have to take another examination and adorn his name with still more letters before he could hope to be considered for a vacancy in any executive council area. Have we not had enough of what Professor Harris has called the tawdry diplomas that clutter up medical education?

Some of your correspondents complain that our prestige has slipped. That is partly our own fault. Others say that our pride must be restored. How can it be restored by pretending that something correctly described as *general* can also be a *specialty*?—I am, etc.,

Rugby. W. J. BOLD.

SIR,—While wholeheartedly supporting the idea of a General Practitioner College, the practitioners in the Winchester area have anticipated its need by setting up an association for general practitioners, which is now known as the E.C.1 Club. The objects of this club are: to encourage "the meeting of one's colleagues socially"; the exchange of views and opinions on matters clinical; the discussion and ventilation of problems and topics affecting a general practitioner.

The first half-hour of our meeting is covered by the phrase "reconnaissance and refreshment," which gives everyone a chance to meet colleagues from outlying areas. We have no formal lecture on an abstruse subject of rare pathology, but a consultant guides, referees, and sums up our discussions on any subject. The advantage of this mode of procedure is that we, knowing the gaps in our knowledge, have the choice of any consultant (who is always a man of experience) rather than a lecturer chosen for us by our consultant colleagues. We find practitioners travelling from as far away as 25 miles to attend a meeting, which shows that some such society or club of this nature is needed.—We are, etc.,

P. A. T. LOWDEN.
Winchester. GEORGE SWIFT.

Reducing Dislocated Hip

SIR,—May I suggest what I think is an improvement on the current method of reducing a posterior dislocation of the hip-joint?

Anaesthetize as usual, but, instead of laying the patient on the floor, put him on his back on the operating table in the usual way, flex the hip and the knee to a right angle, and put the limb in the neutral position as regards rotation. An assistant, standing on the side of the normal hip, grasps the iliac crests and fixes the pelvis. The operator, facing the patient's feet, inserts his appropriate shoulder into the right angle made by the knee flexed on the thigh. To do this he has to bend forward, perhaps 30 degrees. He grasps the leg by the ankle, merely to preserve the right angle, and straightens his spine.

I have had the chance of doing this four times, and in each case the reduction was instantaneous and the effort required minimal. Even a surgeon who thinks he has a "disk" need have no fear on this score. I can think of no method less likely to inflict further damage to the torn capsule, the preservation of whose blood supply is so vital if avascular necrosis of the head of the femur is to be prevented.—I am, etc.,

Birmingham, 29. J. B. BAIRD.

Do You Smoke?

SIR,—Last week I sent a letter personally to every man and woman on the *Medical Register* of the U.K. asking them to help me. I asked them to fill in a very simple form about their smoking habits. I tested the form, earlier this year, by sending it to 200 doctors taken at random from the *Register*; they nearly all answered. This suggests that the form presents no grave difficulties, and the response has emboldened me to cast my bread upon the waters and to appeal to the whole 60,000.

My aim, I need hardly say, is to add up the returns statistically without any regard to individuals. I hope to be able to divide the profession into about four broad categories—heavy smokers, moderate smokers, light smokers, and non-smokers—and then, over the next few years, and with the help of the Registrars-General of the U.K., measure the mortality of each group in relation to the certified causes of death. Cancer of the lung is by no means the whole interest; other points of importance may well emerge.

This, I think, is a new method of approach—looking forwards in a normal population rather than backwards in declared patients. If it succeeds I hope to extend the inquiry; but clearly the medical profession with its close interest in research should come first. May I, therefore, repeat my appeal through your columns? If every doctor, whatever his field of work, will spare only a moment or two this research can be founded on a firm basis and in time give, I believe, firm and important answers.—I am, etc.,

London, W.C.1. A. BRADFORD HILL.

POINTS FROM LETTERS

Examining the Dead

Dr. M. ASTON KEY (Southsea) writes: A clergyman told me some years ago that he began his student days in the faculty of medicine, but afterwards changed to that of theology. While a medical student he won a prize, offered for the best proof of the fact of death, by saying that the absence of the red glow familiar to us all when a hand is held before the light of an electric bulb was the best and simplest evidence. Shortly after this I was hastily called to the bedside of an old lady patient of mine with severe heart disease and who had suddenly expired. All the usual signs of death were present, but I thought I would put this fresh test to the proof. The patient's companion was present also, but neither she nor I could detect any difference between the appearance of the deceased lady's hand and our own when tested by the bulb of a reading lamp.

Clinical Directives in R.A.F.

EX-NATIONAL SERVICE M.O. writes: Having read the astonishing letter by "Squadron Leader" (October 20, p. 973) I could not let it go unanswered. During my period of national service I, like "Medical Officer," was pestered with frequent clinical directives. One directive stated that medical officers should not diagnose a woman as being pregnant unless she had (*a*) amenorrhoea, (*b*) a (palpable abdominal) swelling of the uterus. Presumably the writer of the directive had not heard of the Friedman test. Another directive stated that all medical officers should note that one millilitre was the same quantity as one cubic centimetre of fluid. These are but two examples, and they serve to show the type of mind that constructs these directives for the attention of conscripted medical officers.

Correction.—Mr. PHILIP B. MAIR (Ciba Laboratories, Ltd.) writes: In the *British Medical Journal* (September 8, p. 570) the word "linguet" is used without the usual inverted commas. It is, however, our registered trade mark.

Obituary

P. L. SUTHERLAND, D.Sc., M.B.

Dr. P. L. Sutherland, emeritus professor of forensic medicine at Leeds University, died on October 27. He was 71 years of age.

Peter Lindsay Sutherland was born and educated in Glasgow. After graduating in medicine with honours in 1903, he specialized in bacteriology and in due course was awarded the degree of D.Sc. for his published work in this field. He was for a time senior assistant bacteriologist to the City of Glasgow, but his principal post, which he held for over 35 years, was that of pathologist to the West Riding County Council. He created the laboratory service in that area, and at first he had only one assistant; in consequence he himself had to undertake much of the technical work. By the time of his retirement in 1945, however, he had established one of the principal laboratories of its kind in this country.

Although primarily interested in bacteriology, to the field of which he made several contributions, his professional interests included pathology and, in particular, forensic pathology. It would seem that his introduction to the latter was by force of circumstances rather than choice. His post entailed the performance of necropsies, and it was characteristic of him that, having to undertake these duties, he applied his talents to them with the same success that he had achieved in bacteriology. He was a founder member of the British Association of Forensic Pathologists, and it was not long before he was recognized by the Home Office as a consulting pathologist. In due course he had gained an unrivalled reputation as a medical jurist, and inevitably his was the responsibility for the medical investigations arising out of murders and other major crimes in the area. The thoroughness of his investigations, the reliability of his opinion, and the transparent quality of his honesty and fair-mindedness gained universal respect. By virtue of these attributes he was a formidable witness to cross-examine.

It was no occasion for surprise, but, on the contrary, one on which to congratulate the University of Leeds, when in 1938 he was invited to occupy the chair of forensic medicine, recently vacated by the late Professor Maxwell Telling. It is a matter for regret that Professor Sutherland's other duties left him no opportunity to publish any of his work in the field of forensic medicine.

C. J. P. writes: The present melancholy task is a privilege which was gladly accepted, because it is an opportunity to pay Professor Sutherland tribute and to express gratitude for his kindness. Peter Sutherland was deceptive in physical appearance. Far from robust, he had a remarkable capacity to endure hard conditions. Even the keenest of winds, on the bleakest of Yorkshire moors, where not infrequently his forensic duties took him, left him unperturbed and apparently unharmed. His appearance may have misled strangers to think him a solemn man of sombre mood. On the contrary, he had a keen and pawky sense of humour, which has been inherited by his doctor son, and he was one of those delightful men whose whole appearance is transformed by their smile. I have never heard an adverse word of comment on him, but there are many who have warm praise for him. Even those who have met him only once or twice succumbed to the spell of his personal charm and were prompt to realize that this was no ordinary man. Not least of his qualities was his generosity. He was at all times ready to give freely of his time, experience, and wide knowledge to those who consulted him. He was particularly kind to his juniors. His successor in the chair will always have good reason to be grateful to him, because he lent him his considerable lecture material, without condition upon the use made of it.

Peter Sutherland can have had no enemies. He had many friends, and those of his acquaintance who have an abiding regard and respect for him are innumerable.

Despite the heavy toll of his many duties Peter Sutherland found time for leisure. In part it was given to the advancement of his knowledge of German, of which language he had a love and of which he was no mean scholar, even if, at times, his reading was of works which included detective fiction, in preference to Goethe. He delighted in the art of fly fishing, which he practised in many waters, both locally and on his native heath. No small part of his time was given to the craft of freemasonry, of which order he was a prominent member.

CHARLES DONOVAN, M.D.

Lieutenant-Colonel, I.M.S.

We record with regret the death on October 29 of Colonel Charles Donovan, of the Indian Medical Service. He was 88 years of age. For the following appreciation we are indebted to Sir Rickard Christophers:

It is with regret that I have heard of the death of Colonel Charles Donovan, whose name is familiar to all students of tropical medicine as the joint discoverer of the causative organism of kala-azar, for long familiarly known as the Leishman–Donovan bodies. It was many years ago—to be exact, in 1904—when I first got to know Colonel (then Major) Donovan shortly after his discovery. In that year, having recently joined the Service, I had been sent to Madras to study these bodies and well remember Colonel Leslie, my chief, saying, "Donovan is Irish, so stand up to him and it will be all right." However, the advice was not necessary, as no one could have been more friendly and frank about every detail of his discovery. He helped me in many ways, gave me the free run of his wards, and he and Mrs. Donovan were so kind and hospitable that I have never forgotten those days in Madras. Donovan was then one of the three senior physicians at the Madras General Hospital, one of the most remarkable hospitals I have ever in my experience met with, being a perfect storehouse of material in tropical diseases, with men in charge, including Donovan himself, whose keenness and medical experience were an education to meet. Donovan was greatly beloved and almost worshipped by his students, and his was indeed a very dynamic and original personality. It was no accident that he discovered the bodies, for he had an unusually inquiring mind. Apart from medicine and teaching his students, Donovan had many interests. A quite serious hobby was the Lepidoptera, on which I think he published papers. He was a competent amateur artist, and at the time I refer to was struggling to get to his satisfaction some of the gorgeous sunset effects of Madras. He was very direct and outspoken, but one never minded, because there was always friendliness behind it.

A. MISSIROLI, M.D.

Professor Alberto Missiroli, director of the section of parasitology, Istituto Superiore di Sanità Pubblica, died in Rome on July 18, 1951, aged 68. Missiroli was born at Cervia, near Ravenna, and graduated in medicine at Bologna University in 1908, subsequently studying hygiene and bacteriology at the Institute of Hygiene, Siena. In 1910 he took part in a campaign against cholera in Puglia, and shortly afterwards he was transferred to Sardinia, where he established and directed the bacteriological laboratory at Sassari. During this period he studied the biology of the cholera vibrio and the problem of brucellosis in Sardinia. In 1914 he joined the public health laboratory in Rome, where he carried out biological and serological studies on influenza and on amoebic and bacillary dysentery. Three years later he assisted Professor B. Gosio, director of the laboratory, in establishing the School of Rural Hygiene and Malaria Prevention at Nettuno, on the coast at the western edge of the Pontine Marshes.

In 1924 Dr. L. W. Hackett, of the International Health Division of the Rockefeller Foundation, was sent to Italy to organize a centre for the study and prevention of malaria, and Missiroli was selected as director of the Malaria Experiment Station which was established in Rome in the following year in collaboration with the Italian Government. From then onwards he devoted himself exclusively to the study of the epidemiology and control of malaria. Hackett and Missiroli were the first to use Paris green as a mosquito larvicide on a wide scale. After preliminary trials in Sardinia this method was successfully applied in many of the malarious parts of Italy. A detailed account of the epidemiological studies undertaken and the various control measures used during this period was contained in a comprehensive monograph published in 1934. Missiroli's close association with Hackett was emphasized by the latter in his book *Malaria in Europe*, published in 1937, in which he wrote; "To Professor Missiroli I stand in a special relationship. We have collaborated for such a long time that I hardly know which are his ideas and which my own."

Working with Hackett and with Professor E. Martini, of Hamburg, Missiroli followed up and amplified the researches of Falleroni on the different biological races of *Anopheles maculipennis*, an ecological study which shed new light on a number of hitherto obscure problems, such as anophelism *sine* malaria and the spontaneous regression of malaria in certain regions of Europe. He was also associated with Sir Rickard Christophers in the writing of a monograph on *Housing and Malaria*, published in 1933, which represents a most valuable contribution to the study of mosquito bionomics.

When D.D.T. was introduced as an insecticidal agent at the close of the second world war, Missiroli was quick to recognize its potentialities for the control of malaria. The Experiment Station in Rome had by that time been absorbed as a section of the Istituto Superiore di Sanità Pubblica, with Missiroli as its chief. Malaria had once more become a major problem in the Pontine Marshes as the result of damage to drainage works by military operations, and he and his colleagues directed all their energies to the destruction of adult mosquitoes with D.D.T. and other insecticides, with the result that the disease was completely eliminated in that area. Similar methods were thereafter introduced into every malarious region in the country, with such success that before his death Missiroli had attained the objective which he had pursued for so many years—namely, the virtual elimination of malaria from Italy by measures directed against the insect vector of the disease. He will be remembered not only for his outstanding achievements in the field of malariology, but also for the unvarying courtesy and modesty which endeared him to all with whom he came in contact.—G. C.

EMILE BRUMPT, M.D., D.Sc.

By the death of Professor Emile Brumpt on July 7, 1951, the scientific world has lost one of the most renowned of the great figures who dominated the field of parasitology during the first half of this century.

Born in Paris on March 7, 1877, Emile Brumpt was educated at the Sorbonne and the faculty of medicine, University of Paris, from which he received the degrees of D.Sc. (1901) and M.D. (1906). From the very beginning of his career he devoted himself to medical zoology, in which he was initiated by his teacher Raphaël Blanchard, under whom he served successively as assistant and lecturer, until (in 1919) he succeeded him as professor in the faculty of medicine and director of the parasitological laboratory, posts which he held till his retirement in 1948. From modest beginnings Brumpt raised the laboratory to the status of an institute, which became a world-famous school and research centre of parasitology. In the course of more than 50 years Brumpt made numerous important contributions to this field of study. To him belongs the merit of being the first to demonstrate in trypanosomes a life-cycle with an alternation of vertebrate and invertebrate hosts. He also considerably advanced our knowledge of the methods of transmission of pathogenic micro-organisms by arthropod vectors, and introduced xenodiagnosis for the detection of Chagas's disease. But his most outstanding achievement is undoubtedly his *Précis de Parasitologie*. Started in 1910 and now in its sixth edition (1949), this comprehensive treatise has for many years been an indispensable source of reference to students and research workers throughout the world. In the course of his life Brumpt travelled widely in search of parasitological material, for the discovery of which he relied not only on his extensive knowledge but also on his unerring hunter's instinct. Brumpt also founded and edited the well-known *Annales de Parasitologie Humaine et Comparee*.

Brumpt's work was recognized by many awards and honours, both in France and even more in foreign countries. Suffice it to say that he was Vice-President of the French Academy of Medicine, member of the Academy of Colonial Sciences, of the Academy of Veterinary Medicine, and president of several learned societies, including the Société de Pathologie Exotique. He was a foreign member of a number of academies and honorary fellow of many scientific societies (including the Royal Society of Tropical Medicine and Hygiene) both in the old and new worlds. It is, however, a sad reflection on the fate of "a prophet in his own country" that—though thrice heading the list of candidates—he was not accorded the supreme honour of being elected to the Institut de France (the equivalent of our Royal Society).

Shortly after the last war Brumpt was stricken by an illness which confined him to his bed to the end of his days. His last public appearance was in 1948, when the

French medical world celebrated his scientific jubilee and collected funds for a "Prix Emile Brumpt," to be awarded to outstanding workers in parasitology and tropical medicine.—C. A. H.

Group Captain ROBERT ANDREW GEORGE ELLIOTT, until recently medical superintendent of Stoke Mandeville Ministry of Pensions Hospital, Aylesbury, died at his home on October 5. He graduated M.B., B.Ch., B.A.O. from Trinity College, Dublin, in 1913, having completed his clinical studies at Dr. Steevens's Hospital in that city. He served in the 1914 war in the R.F.C. at Uxbridge Hospital, Egypt, and Aden. On his retirement from the R.A.F. he was appointed medical superintendent of the Ministry of Pensions Hospital, Walton, Liverpool, and not long afterwards medical superintendent of Stoke Mandeville Ministry of Pensions Hospital, now taken over by the Ministry of Health. His medical career was mainly occupied in an administrative capacity—for which he was eminently suited. He upheld all that was best in his profession and achieved efficiency at all times. Ready at any time to give helpful advice should any difficulty arise, he was loyal to his staff, and in return commanded their complete trust and respect. His loss is felt by all his many lifelong friends, who extend their sincere sympathy to his widow and son.—G. S.

Medico-Legal

AN ALCOHOLIC WIFE

[FROM OUR MEDICO-LEGAL CORRESPONDENT]

Mr. Commissioner Blanco White recently had before him in the Divorce Court[1] a petition for divorce by a husband alleging constructive desertion by his wife on the ground that she had driven him to leave her by her drinking habits.

To constitute desertion within the meaning of the Matrimonial Causes Act, 1950, there must be, as in the case of the Act of 1937 which the Act of 1950 replaces, both the fact of separation and an intention of the deserting party to bring the matrimonial cohabitation to an end. In the case of *Williams* v. *Williams*[2] it was established by the Court of Appeal that as a matter of law a certified lunatic could not have such an intention, and that accordingly, although the husband in that case had originally deserted his wife, the period of desertion ceased when he was certified.

Mr. Commissioner Blanco White found in the case before him that the wife had had no intention to drive her husband out of the home, and that therefore she had not deserted him.

He added that, although the husband knew his wife was an alcoholic and must have been warned by her doctors, he allowed her while on holiday in France to drink wine and champagne, cocktails and martinis, which was regrettable behaviour on his part. But this did not affect the question of the wife's intention to drive him away except by making attacks of acute alcoholism more likely.

As a matter of law, provided the fact of separation and the intention of the guilty party to bring cohabitation to an end are present, it does not matter which party goes and which stays behind. It is the intention of the guilty party which is the crux of the matter. And it is paradoxical that a condition of chronic alcoholism or insanity, which at first sight would appear to justify the innocent party in leaving the other, far from facilitating divorce on the ground of desertion, will in all probability make it impossible by depriving the deserter of the necessary intention.

[1] *Evening Standard*, June 18.
[2] [1939], p. 365.

Universities and Colleges

UNIVERSITY OF CAMBRIDGE

The Vice-Chancellor (Sir Lionel Whitby) and Professor A. Leslie Banks have been appointed two of the University's representatives at the Conference of Home Universities to be held on December 14 and 15.

In Congregation on October 27 Dr. E. D. Adrian, O.M., P.R.S., Master of Trinity College, was appointed a Manager of the E. G. Fearnsides Fund until December 31, 1953.

UNIVERSITY OF GLASGOW

On October 26 the William Mackenzie Medal was awarded to Mr. H. B. Stallard for valuable contributions to ophthalmology.

UNIVERSITY OF LONDON

The Semon Lecture for 1951 entitled "Articulatory Defects in Aphasia" will be delivered by Dr. Macdonald Critchley, Dean of the Institute of Neurology, the National Hospital, Queen Square, London, W.C., at the Royal Society of Medicine, 1, Wimpole Street, London, W., on Thursday, November 1, at 5 p.m. The lecture is addressed to students of the University and to others interested in the subject. Admission is free, without ticket.

Dr. Ruth Bowden has been appointed to the University Chair of Anatomy tenable at the Royal Free Hospital School of Medicine, from October 1.

Dr. Richard John Harrison has been appointed Head of the Department of Anatomy at London Hospital Medical College, and the title of Reader in Anatomy has been conferred on him in respect of this post.

The following candidates have been approved at the examinations indicated.

ACADEMIC POSTGRADUATE DIPLOMA IN CLINICAL PATHOLOGY.—J. G. Azzopardi, R. Cassel, I. M. Chaudhri, O. L. C. Cookson, R. D. Eastham, G. M. Edington, P. I. A. Hendry, Roberta G. Horsley, Thelma M. Hunt, G. Kemble Welch, Margaret K. B. Knox, J. Kohn, K. A. Monsur, R. Mortimer, A. M. Mukherjee, P. O'Connell, B. D. P. Rao, G. K. Tiagi.

EXTERNAL DIPLOMA IN CLINICAL PATHOLOGY.—F. E. Aaron, M. C. Botha, A. J. E. Brafield, F. G. L. Brafield, N. J. H. Davidson, M. Rahman, R. C. Woodcock.

The following candidates at the London School of Hygiene and Tropical Medicine have been approved at the examination indicated:

ACADEMIC POSTGRADUATE DIPLOMA IN PUBLIC HEALTH.—J. G. S. Holman, F. G. Sembeguya.

UNIVERSITY OF LIVERPOOL

The following candidates have been approved at the examination indicated:

FINAL M.B., Ch.B.—*Part I:* Cicely G. Brenner, T. R. W. Cowell, W. Deighton, M. A. R. Eslick, C. D. Fisher, Winifred M. Jones, J. H. Leavesley, I. V. Lishman, Eleanor C. Lloyd, M. Pearson, D. F. Rice, Margaret J. Ringham, Eileen M. Shuttleworth, A. M. Stewart, T. J. Walsh, Mary B. Woodfield, P. J. J. Wren.

ROYAL COLLEGE OF SURGEONS OF EDINBURGH

At the annual meeting of the College held on October 17, the following officers were elected for the ensuing year: *President*, Professor Walter Mercer. *Vice-President*, Mr. W. Quarry Wood. *Secretary and Treasurer*, Mr. J. J. Mason Brown. *Representative on the General Medical Council*, Sir Henry Wade. *Conservator of Museum*, Mr. J. N. J. Hartley. *Convener of Museum Committee*, Mr. John Bruce. *Librarian*, Dr. Douglas Guthrie.

The Liston Victoria Jubilee Prize, which is awarded to the Fellow of the College who has made the greatest practical contribution to surgery in the previous four years, was presented to Dr. John Gillies.

The following candidates, having passed the requisite examinations, were admitted Fellows: J. Broadfoot, N. A. G. Covell, P. K. Devi, P. P. Goel, W. R. Grant, J. W. L. Haddon, K. M. Kamodia, T. W. H. Lamont, B. T. le Roux, R. Misra, R. D. Muckart, K. McLay, R. Prasad, T. K. Whaley, R. J. Wilson.

ROYAL COLLEGE OF PHYSICIANS OF IRELAND

Dr. Leonard Abrahamson has been re-elected President of the College and Dr. G. T. O'Brien Vice-president.

INFECTIOUS DISEASES AND VITAL STATISTICS

We print below a summary of Infectious Diseases and Vital Statistics in the British Isles for the week ending October 20 (No. 42).

Figures of notified *cases* are for: (*a*) England and Wales (London included). (*b*) London (administrative county). (*c*) Scotland. (*d*) Northern Ireland. (*e*) Eire. Figures of *births and deaths* and of *deaths* recorded under each disease are for: (*a*) The 126 great towns in England and Wales (London included). (*b*) London (administrative county). (*c*) The 16 principal towns in Scotland. (*d*) The 10 principal towns in Northern Ireland. (*e*) The 13 principal towns in Eire. A dash — denotes no cases; a blank space denotes disease not notifiable or no return available.

The table is based on information supplied by the Registrars-General of England and Wales, Scotland, Northern Ireland and Eire, the Ministry of Health and Local Government of Northern Ireland, and the Department of Health of Eire.

Disease	1951 Week Ending October 20					1950 Corresponding Week					1942–50 England & Wales Corresponding Week		
	(*a*)	(*b*)	(*c*)	(*d*)	(*e*)	(*a*)	(*b*)	(*c*)	(*d*)	(*e*)	Highest	Median	Lowest
Diphtheria	33	3	12	—	1	42	6	17	1	3	957	278	42
Deaths	1	—	—	—	—	—	—	—	—	—			
Dysentery	162	38	48	2	1	169	15	111	—		391	169	46
Deaths		—		—			—		—				
Encephalitis, acute	5	—	—	—		3	1	—	—		4	1	—
Deaths		—			—		—			—			
Erysipelas			21	—	12			28	6	3			
Food-poisoning	88	6		—		138	6		1				
Infective enteritis or diarrhoea under 2 years				16	41				27	28			
Deaths	5		4	—	1	10		1	1	—			
Measles*	1,809	71	100	135	156	5,876	366	140	56	114	6,807	2,385	441
Deaths		—	—	—	—		—	—	1	—			
Meningococcal infection	36	3	17	1	2	31	4	18	1		64	36	24
Deaths		—	—				—	1					
Ophthalmia neonatorum	41	5	11	1	2	37	1	9	—		91	68	37
Pneumonia, influenzal	424	18	5	1		432	23	3	1		661	518	317
Deaths (from influenza)†	18	2	1	—	—	12	2	—	—	1			
Pneumonia, primary			157					141					
Deaths	151	25		7	12	161	23		10	4			
Poliomyelitis, acute:													
Paralytic	67	—	19	5	4	278	17	29	5	6	414	30	14
Non-paralytic	52	2											
Deaths§	3	—			—	12	2			—			
Puerperal fever			11					4					
Puerperal pyrexia‖	245	33	5	2		72	3	8	1		162	134	72
Scarlet fever	1,135	84	260	32	43	1,227	81	193	46	81	3,328	1,768	1,048
Deaths		—	—	—	—		—	—	—	—			
Smallpox	—	—	—	—		—	—	—	—				
Deaths				—					—				
(1) Tuberculosis, respiratory			151	34				146	29				
(2) Tuberculosis, non-respiratory			24	5				28	4				
(1) Deaths	158	29	17	2	3	142	14	23	8	7			
(2) Deaths		3	2	—	2		2	2	3	1			
Typhoid fever	3	—	—	—	—	3	—	1	2	1			
Deaths¶	—	—	—	—		—	—	—	—				
Paratyphoid fever	81	—	1 (B)	—	1 (B)	13	3	—	2	1 (B)	27	17	7
Whooping-cough	1,516	85	317	37	72	2,674	251	303	35	54	2,674	1,087	834
Deaths	—	—	1	—	1	5	—	—	—	—			
Deaths (0–1 year)	176	23	25	14	19	213	30	38	13	17	358	322	213
Deaths (excluding stillbirths)	4,450	704	510	107	146	4,447	716	565	132	146	4,447	4,085	3,818
Annual death rate (per 1,000 persons living)			10·5					11·4					
Live births	6,340	1,060	807	198	380	6,797	1,126	886	200	410	8,660	6,797	5,847
Annual rate per 1,000 persons living			16·5					17·8					
Stillbirths	171	33	12			179	23	21			295	200	172
Rate per 1,000 total births (including stillborn)			15					23					

* Measles not notifiable in Scotland and returns are approximate. † Includes primary form for England and Wales, London, and N. Ireland. § The number of deaths from poliomyelitis and polio-encephalitis for England and Wales, and London (administrative county), are combined. ‖ Includes puerperal fever for England and Wales and for Eire. ¶ Deaths from paratyphoid fever are combined with those from typhoid fever.

Vital Statistics

Death Rates in Second Quarter

According to the Registrar-General's return for the quarter ending June 30, the death rate in England and Wales in that quarter from influenza was 40 per million population. In the corresponding quarter in 1950 it was 84. Death rates for other infectious diseases (with rates for the 1950 corresponding quarter in parentheses) were as follows: whooping-cough 9 (9), pneumonia 416 (385), acute poliomyelitis and polio-encephalitis 4 (6). The death rate from enteritis and diarrhoea under 2 years was 1.2 per thousand live births, as compared with 1.7 in 1950.

Road Accidents

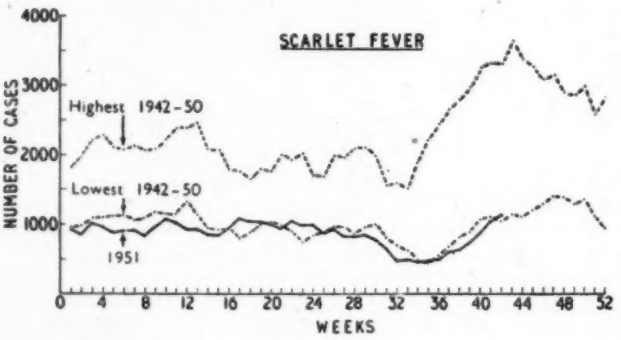
Road accidents during August totalled 21,819. 470 persons were killed and 21,349 injured, 5,349 seriously. The position was rather worse than in August, 1950, when total casualties were 20,951, but not quite so serious as in August, 1938 (the last pre-war August for which figures are available), when the total was 22,858.

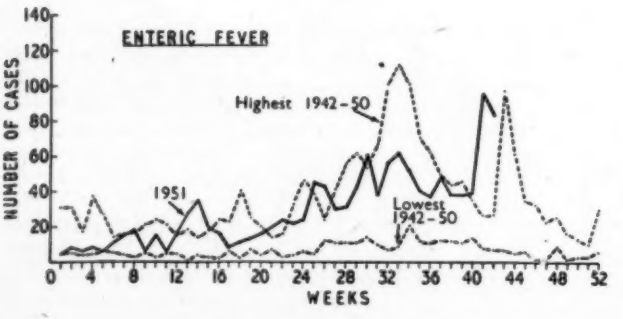
Week Ending October 27

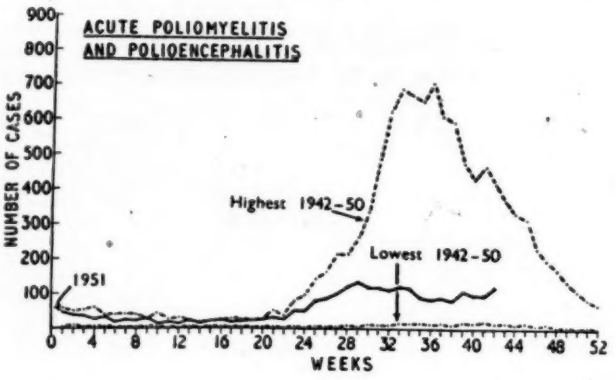
The notifications of infectious diseases in England and Wales during the week included: scarlet fever 1,116, whooping-cough 1,441, diphtheria 35, measles 1,962, acute pneumonia 419, acute poliomyelitis 96, dysentery 151, paratyphoid fever 29, and typhoid fever 4.

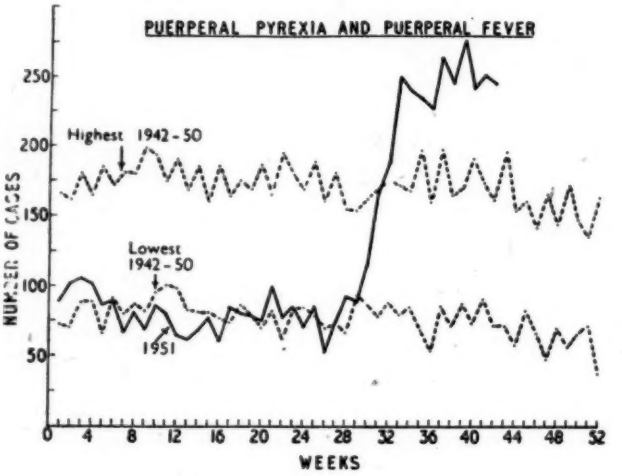
Graphs of Infectious Diseases

The graphs below show the uncorrected numbers of cases of certain diseases notified weekly in England and Wales. Highest and lowest figures reported during the nine years 1942–50 are shown thus - - - - - - -, the figures for 1951 thus ————. Except for the curves showing notifications in 1951, the graphs were prepared at the Department of Medical Statistics and Epidemiology, London School of Hygiene and Tropical Medicine.









Infectious Diseases

Increases were recorded in the number of notifications of measles 161, scarlet fever 103, dysentery 44, acute poliomyelitis 25, and acute pneumonia 29 in England and Wales during the week ending October 20, while the only decrease was 96 for whooping-cough.

Only small changes occurred in the local incidence of scarlet fever; the largest was a rise of 26 in Northumberland. The increase in the incidence of measles was mainly in the North Midland counties; the largest rise was 64 in Staffordshire. The notifications of diphtheria were 8 fewer than in the preceding week; the largest variations in the local returns were falls in Northumberland 7, Warwickshire 3, and a rise of 3 in Lancashire. The trends of whooping-cough showed the usual fluctuations; the largest rise was Staffordshire 25, and the largest fall was Yorkshire West Riding 44.

Of the 81 cases of paratyphoid fever 54 were notified in Yorkshire West Riding (Bradford C.B. 23, Halifax C.B. 8, Keighley M.B. 5, Huddersfield C.B. 4, Brighouse M.B. 4). Of the remaining 27 cases 13 were notified in Lancashire; these cases were spread through 8 administrative areas.

The number of notifications of paralytic poliomyelitis was 13 more and the number of notifications of non-paralytic poliomyelitis was 12 more than in the preceding week. With four exceptions the numbers of notifications were the highest during this year. The largest returns during the week were Yorkshire West Riding 21 (Sheffield C.B. 8), Lancashire 13 (Liverpool C.B. 4), Somersetshire 7 (Yeovil R.D. 4), Devonshire 6 (Plymouth C.B. 3), Derbyshire 6 (Chesterfield M.B. 3), Northumberland 6 (Newcastle-upon-Tyne C.B. 4), Staffordshire 6.

The largest centres of dysentery during the week were London 38 (Islington 16), Lancashire 22, Kent 21 (Malling R.D. 15), Middlesex 18 (Edmonton M.B. 11).

Medical News

A Book for Christmas

50 Years of Medicine, containing in book form articles surveying the progress of medicine and surgery during the first half of this century which appeared in a special number of the *Journal* last year, is again available. Very pleasantly brought out, and handsomely bound, not only does it look impressive but the information it gathers together is useful and enlightening, and is of interest to both lay and medical readers. Over 300 pages of text, a complete index, and 32 illustrations make this a valuable book to have, and, incidentally, an excellent Christmas present. Costing 15s., *50 Years of Medicine* may be obtained through booksellers, or direct (post free) from the Publishing Manager, B.M.A. House, Tavistock Square, London, W.C.1.

Smoking and Lung Cancer

Investigations into the possible association between tobacco-smoking and carcinoma of the lung are being continued by Professor A. Bradford Hill and Dr. W. R. S. Doll, whose paper on the subject appeared in the *Journal* of September 30, 1950 (p. 739). Professor Bradford Hill is sending (see our correspondence columns this week, p. 1157) a questionary to all registered medical practitioners asking them to record briefly their smoking habits. These particulars can be related to mortality statistics recorded by the Registrar-General. The replies will be strictly confidential, and it is hoped that every doctor will give the information required for this important piece of research.

Guide to Postgraduate Scholarships

The British Commonwealth Scientific Office (London) has just published a useful guide to all medical and other scientific postgraduate scholarships and fellowships offered in Britain and other Commonwealth countries to open competition. The great majority are awarded in the United Kingdom, but Canada, Australia, New Zealand, India, and Pakistan are also covered. *Inter-Commonwealth Postgraduate Scholarships in Science* is obtainable from the Stationery Office and usual agencies, price 5s.

A Great Embryologist

The centenary of the birth of Francis Maitland Balfour, the biologist, falls on November 10. He was the younger brother of A. J. Balfour, Prime Minister from 1902 to 1905 and later Earl of Balfour. F. M. Balfour had a distinguished student career at Cambridge and was appointed lecturer in animal pathology there in 1876. Two years later he was elected F.R.S. In 1880–1 appeared his great two-volume *Treatise on Comparative Embryology*. This was a masterly digest of all previous work (including Balfour's own classical contributions) upon which modern embryology is based. Early in 1882 a special professorship of animal morphology was instituted for him at Cambridge. Later that same year Balfour started out on a mountaineering holiday in the Swiss Alps and met a tragic end, apparently on July 19, when both he and his guide fell while on the Aiguille Blanche de Peuteret. Balfour was held in great esteem by his teacher, Michael Foster, who wrote his obituary in the *British Medical Journal* and his biography in the *Dictionary of National Biography*.

A Film on the Care of Old People

Dr. Trevor H. Howell has had made a silent film lasting about 20 minutes which traces the history of one rheumatic patient from the moment she is seen by her own doctor, through her admission to hospital and transfer to a half-way house, to her final discharge home. Great emphasis is laid on physiotherapy and occupational therapy, and on the value of the half-way house. On October 31, when the film was shown at the Hammer Theatre, Dr. Howell personally gave a running commentary which was very effective, and which the film needs. With it the film could be a useful introduction for both lay and medical (student) audiences to some of the special problems of a geriatric unit.

Doctor in the Government

It was announced on October 31 that Dr. Charles Hill, M.P. for Luton and former Secretary of the B.M.A., has been appointed Parliamentary Secretary to the Ministry of Food.

British Cardiac Society

The autumn meeting of the British Cardiac Society was held in London on October 19 under the chairmanship of Dr. J. H. Wright. Both the morning and afternoon sessions were devoted to short communications, including papers on mitral valvulotomy, the results of ligation of the patent ductus arteriosus, the bronchodilator action of khellin, clinical and cardiographical aspects of ambiguous cases of cardiac infarction, and the F.933 test in hypertension. A dinner was held at the Savoy Hotel.

Age of Marriage in Northern Ireland

The marriage age in Northern Ireland is to be brought into line with that in Great Britain. The Government has introduced a Bill by which, in future, marriages of persons under 16 will be void. Hitherto the age has been 12 in respect of girls and 14 for boys.

Emergency Bed Service: Applications and Admissions

During the seven days ending November 5 the number of applications made by doctors to the London Emergency Bed Service for admission of patients was 960, of whom 88.02% were admitted.

COMING EVENTS

Society of Medical Officers of Health

The annual dinner of the Society of Medical Officers of Health will be held at the Piccadilly Hotel, London, W., on Thursday, November 22, at 7 for 7.30 p.m., when it is hoped that the newly appointed Minister of Health and Secretary of State for Scotland will be able to attend.

Stephen Paget Memorial Lecture

The twentieth Stephen Paget Memorial Lecture of the Research Defence Society will be delivered by Sir Wilson Jameson at the London School of Hygiene and Tropical Medicine, Keppel Street, Gower Street, W.C., on Thursday, November 22, at 5.30 p.m. His subject is "Research and Administration—A Profitable Partnership?" The annual general meeting of the Society will be held after the lecture.

British Empire Cancer Campaign

The annual meeting of the Nottinghamshire Council of the British Empire Cancer Campaign will be held at the Y.W.C.A. Hall, Shakespeare Street, Nottingham, on Tuesday, November 13, at 3 p.m., when Lord Horder will deliver an address. All medical practitioners are invited to attend.

West Midlands Physicians Association

The third meeting of the West Midlands Physicians Association will be held at Queen Elizabeth Hospital, Birmingham, on Saturday, November 17, at 11 a.m. There will be case presentations during the morning and general business and scientific communications in the afternoon.

Medical Films

Dr. Brian Stanford will give a talk on "Medical Films: Their Cataloguing and Care" to the Medical Section of the Library Association at the British Dental Association, 13, Hill Street, Berkeley Square, London, W.1, on Friday, November 16, at 7 p.m. The meeting is open to all.

Home Safety

A meeting, arranged by the City of Nottingham, the Royal Society for the Prevention of Accidents, and the Royal Institute of Public Health and Hygiene, will be held at the Young Women's Christian Association (Shakespeare Street, Nottingham) on Wednesday, November 28, at 6.15 p.m., when Dr. J. L. Burn will speak on "Home Safety and Health," and Mrs. Winifred E. Duncan on "The Formation of the Nottingham Home Safety Committee." Admission is free, without ticket.

"Outwitting Handicaps"

The Central Council for the Care of Cripples is arranging an exhibition of aids for the disabled in the Goldsmiths' Hall, Foster Lane, London, E.C.2, on November 27 and 28 from 10 a.m. to 6.30 p.m. Admittance will be free.

International Symposium on Yaws Control

The symposium will be held in Bangkok, Thailand, on March 14 to 22, 1952, under the auspices of the World Health Organization in collaboration with the Government of Thailand. During the week following the symposium, a field demonstration and training seminar will be conducted, utilizing the facilities of the W.H.O. Yaws Control Team in that country. All interested in the subject are cordially invited to attend. Copies of the programme will be available about February 1, 1952. Requests for copies should be sent to Dr. Frank W. Reynolds, World Health Organization, Palais des Nations, Geneva, Switzerland. Inquiries may be addressed to any regional office of the World Health Organization or to W.H.O. Headquarters in Geneva.

SOCIETIES AND LECTURES

A fee is charged or a ticket is required for attending lectures marked ●. Application should be made first to the institution concerned.

Monday

HUNTERIAN SOCIETY.—At Society of Apothecaries of London, Black Friars Lane, Queen Victoria Street, London, E.C., November 12, 8.30 p.m., *"That this Age of Speed has Outrun Human Adaptation,"* proposed by Dr. Macdonald Critchley and the Right Rev. the Lord Bishop of Pontefract, opposed by Air Vice-Marshal J. M. Kilpatrick and Mr. Raymond Mays. Discussion.

MEDICAL SOCIETY OF LONDON, 11, Chandos Street, Cavendish Square, London, W.—November 12, 8.30 p.m., *"Newer Antibiotics,"* discussion to be introduced by Professor Robert Knox and Dr. K. S. MacLean.

●POSTGRADUATE MEDICAL SCHOOL OF LONDON, Hammersmith Hospital, Ducane Road, W.—November 12, 4 p.m., *"Some Practical Aspects of Immunization,"* by Dr. H. Stanley Banks.

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—November 12, 5 p.m., *"Inflammatory Affections of the Fundus,"* by Professor Arnold Sorsby.

Tuesday

CHELSEA CLINICAL SOCIETY.—At South Kensington Hotel, 41, Queensgate Terrace, London, S.W., November 13, 8.30 p.m., *"The Origin and History of the Order of St. John of Jerusalem (illustrated with cinematograph film),"* discussion to be opened by Lord Wakehurst, Prior of the Order.

●INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—November 13, 5.30 p.m., *"Urticaria, Dermographism, Physical Allergy,"* by Dr. E. J. Moynahan.

LONDON UNIVERSITY.—At London School of Hygiene and Tropical Medicine, Keppel Street, Gower Street, W.C., November 13, 5 p.m., *"Trends of Opinion about the Public Health, 1901–1951. 3.—Hopes and Fears after the First World War,"* Heath Clark Lecture by Professor J. M. Mackintosh.

ROYAL COLLEGE OF PHYSICIANS OF LONDON, Pall Mall East, S.W.—November 13, 5 p.m., *"The Selection of Medical Students,"* Bradshaw Lecture by Dr. D. H. Brinton.

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—November 13, 5.30 p.m., lecture to nurses and ophthalmic auxiliaries, by Miss Iris Kane.

WEST END HOSPITAL FOR NERVOUS DISEASES, 40, Marylebone Lane, London, W.—November 13, 5.30 p.m., *"Vascular Diseases of Central Nervous System,"* clinical demonstration by Dr. T. Rowland Hill.

Wednesday

BRITISH POSTGRADUATE MEDICAL FEDERATION.—At London School of Hygiene and Tropical Medicine, Keppel Street, Gower Street, W.C., November 14, 5.30 p.m., *"Folic Acid and Vitamin B_{12} in the Metabolism of Micro-organisms,"* by Dr. D. D. Woods.

●DAVIDSON CLINIC, 58, Dalkeith Road, Edinburgh.—November 14, 8 p.m., *"Psychology and Politics."* by Dr. W. P. Kraemer.

GLASGOW UNIVERSITY MEDICO-CHIRURGICAL SOCIETY.—At Geography Lecture Theatre (N.E. corner of Medical Quadrangle of University), November 14, 7.30 p.m., *"The Evolution of the Modern Concepts of Disease,"* by Sir Henry Cohen.

HARVEIAN SOCIETY OF LONDON.—At 11, Chandos Street, Cavendish Square, W., November 14, 8.15 p.m., *"Psychiatry and the Law,"* speakers, Mr. John Maud, K.C., and Dr. Lindesay Neustatter.

●INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—November 14, 5.30 p.m., *"Mycology—Trichophyton Infections,"* by Dr. R. W. Riddell.

●INSTITUTE OF UROLOGY.—At St. Paul's Hospital, Endell Street, London, W.C., November 14, 4.30 p.m., *"Prevention of Recurrence in Urinary Calculi,"* by Mr. D. I. Williams.

●LONDON MARRIAGE GUIDANCE COUNCIL, 78, Duke Street, Grosvenor Square, W.—November 14, 6 p.m., *"The Psychology of Marriage Relationships,"* by Dr. Ethel Dukes.

●POSTGRADUATE MEDICAL SCHOOL OF LONDON, Hammersmith Hospital, Ducane Road, W.—November 14, 11.45 a.m., Medical Clinical-pathology Conference.

●ROYAL COLLEGE OF PHYSICIANS OF LONDON, Pall Mall East, London, S.W.—November 14, 5 p.m., *"Cerebral Localization,"* by Dr. W. Russell Brain, P.R.C.P.

ROYAL COLLEGE OF SURGEONS OF ENGLAND, Lincoln's Inn Fields, London, W.C.—November 14, 3.45 p.m., *"The Pathological Aspects of Osteomyelitis,"* Erasmus Wilson Demonstration by Mr. L. W. Proger.

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—November 14, 5.30 p.m., *"The Facies in Disease,"* by Mr. L. H. Savin.

ROYAL FACULTY OF PHYSICIANS AND SURGEONS OF GLASGOW, 242, St. Vincent Street, Glasgow.—November 14, 5 p.m., *"Syndromes of the Thoracic Inlet and Cervico-axillary Canal,"* Finlayson Memorial Lecture by Dr. F. M. R. Walshe, F.R.S.

ROYAL INSTITUTE OF PUBLIC HEALTH AND HYGIENE, 28, Portland Place, London, W.—November 14, 3.30 p.m., *"Modern School Hygiene and the Schoolchild"* (illustrated), by Dr. G. D. Pirrie.

ROYAL SANITARY INSTITUTE, 90, Buckingham Palace Road, London, S.W.—November 14, 2.30 p.m., Symposium on Smallpox. (*a*) *"The First Few Days of the Brighton Outbreak, 1950"*; (*b*) *"Laboratory Diagnosis, with Special Reference to the Brighton Outbreak,"* by Dr. F. O. MacCallum; (*c*) *"Comment,"* by Dr. W. H. Bradley.

Thursday

BRITISH INSTITUTE OF RADIOLOGY, 32, Welbeck Street, London, W.—November 15, 8 p.m., *"Is Your Physics Really Necessary?"* Presidential Address by Professor J. E. Roberts.

DREADNOUGHT SEAMEN'S HOSPITAL, Greenwich, London, S.E.—November 15, 3 p.m., clinical demonstration of surgical cases by Mr. Stephen Power.

EDINBURGH CLINICAL CLUB.—At B.M.A. Rooms, 7, Drumsheugh Gardens, Edinburgh, November 15, 8 p.m., *"Contact Dermatitis,"* by Dr. G. A. G. Peterkin.

HONYMAN GILLESPIE LECTURE.—At University New Buildings (Anatomy Theatre), Teviot Place, Edinburgh, November 15, 5 p.m., *"The Reticuloses from the Point of View of the Radiotherapist,"* by Miss Margaret C. Tod, F.R.C.S.Ed.

●INSTITUTE OF CHILD HEALTH, Hospital for Sick Children, Great Ormond Street, London, W.C.—November 15, 5 p.m., *"Orthopaedic Problems in Infancy,"* by Mr. J. C. R. Hindenach.

LONDON UNIVERSITY.—At London School of Hygiene and Tropical Medicine, Keppel Street, Gower Street, W.C., November 15, 5 p.m., *"Trends of Opinion about the Public Health, 1901–1951. 4.—The Great Depression,"* Heath Clark Lecture by Professor J. M. Mackintosh.

ROYAL ARMY MEDICAL COLLEGE.—At Lecture Theatre, John Islip Street, London, S.W., November 15, 5 p.m., *"The Meaning of Military Uniforms,"* by Mr. James Laver.

ROYAL COLLEGE OF PHYSICIANS OF LONDON, Pall Mall East, S.W.—November 15, 5 p.m., *"The Power of Words,"* Lloyd-Roberts Lecture by Lady Violet Bonham Carter

ROYAL EYE HOSPITAL, St. George's Circus, Southwark, London, S.E.—November 15, 5.30 p.m., *"Post-operative Complications and Their Management,"* by Miss Jean M. Dollar.

ROYAL MEDICAL SOCIETY, 7, Melbourne Place, Edinburgh.—November 15, 8 p.m., Presidents' Annual Dinner. Guest of honour: Sir Henry Cohen.

ROYAL MEDICO-PSYCHOLOGICAL ASSOCIATION.—At 26, Portland Place, London, W., November 15, 2.15 p.m., *"The Assessment of Personality,"* Maudsley Lecture by Sir Cyril Burt.

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.—At London School of Hygiene and Tropical Medicine, Keppel Street, Gower Street, W.C., November 15, 7.30 p.m., laboratory meeting. Various demonstrations will be given.

ST. GEORGE'S HOSPITAL MEDICAL SCHOOL, Hyde Park Corner, London, S.W.—November 15, 4.30 p.m., lecture-demonstration in neurology by Dr. A. S. Paterson.

UNIVERSITY COLLEGE, Gower Street, London, W.C.—November 15, 5 p.m., *"Hospitals and the National Health Service Act,"* lecture on current legal problems by Dr. O. R. Marshall.

WREXHAM AND DISTRICT CLINICAL SOCIETY.—At Wynstay Arms Hotel, Wrexham, November 15, *"Observations on Corneal Biology,"* by Mr. Tudor Thomas.

Friday

BRITISH INSTITUTE OF RADIOLOGY, 32, Welbeck Street, London, W.—November 16, 5 p.m., meeting of medical members.

BRITISH POSTGRADUATE MEDICAL FEDERATION.—At London School of Hygiene and Tropical Medicine, Keppel Street, Gower Street, W.C., November 16, 5.30 p.m., "*Adrenal Hormones and A.C.T.H.*," by Professor F. G. Young, F.R.S.

FACULTY OF RADIOLOGISTS.—November 16, joint meeting of the Therapy Section with the Section of Radiology, Royal Society of Medicine, in connexion with the Royal Cancer Hospital Centenary, 1851–1951. (1) At Royal Cancer Hospital, Fulham Road, London, S.W., 2.30 p.m., demonstrations arranged by Professor D. W. Smithers; (2) At Royal Society of Medicine, 1, Wimpole Street, London, W., 8.15 p.m., papers: "*Cancer of the Breast and the Menopause*," by Professor D. W. Smithers; "*Cancer of the Middle Ear*," by Dr. V. M. Dalley; "*Preliminary Results in the Treatment of Patients with Advanced Carcinoma of the Bladder by 2 MeV Rotation Therapy*," by Dr. J. E. Stapleton; "*The Use of Radioactive Colloidal Gold in the Palliative Treatment of Patients with Pleural Effusions and Ascites Due to Malignant Disease*," by Dr. R. J. Walton.

INSTITUTE OF DERMATOLOGY, Lisle Street, Leicester Square, London, W.C.—November 16, 5.30 p.m., "*Pigmentary Disturbances*," clinical demonstration by Dr. I. Muende.

POSTGRADUATE MEDICAL SCHOOL OF LONDON, Hammersmith Hospital, Ducane Road, W.—November 16, 11.15 a.m., Surgical Clinical-Pathological Conference; 2 p.m., "*Stab Wounds of the Heart*," by Mr. A. T. Andreasen.

ROYAL COLLEGE OF SURGEONS OF ENGLAND, Lincoln's Inn Fields, London, W.C.—November 16, 5 p.m., "*Oral Syphilis*," Webb-Johnson Lecture by Professor R. V. Bradlaw.

ROYAL MEDICAL SOCIETY, 7, Melbourne Place, Edinburgh.—November 16, 8 p.m., "*Idiopathic Ulcerative Colitis*," by Mr. J. D. Cormack.

ROYAL SANITARY INSTITUTE.—At Darlington Town Hall, November 16, 10 a.m., "*Brucellosis: A Disease of Man and Animals*," by Dr. D. J. H. Payne.

SOCIETY OF CHEMICAL INDUSTRY: FINE CHEMICALS GROUP.—At Middlesex Hospital Medical School, Mortimer Street, London, W., November 16, 7 p.m., "*Blood Anticoagulants, with Special Reference to Dextran Sulphate*," by Mr. C. R. Ricketts, Ph.D., and Dr. K. W. Walton.

APPOINTMENTS

AUSTIN, THOMAS A., C.M.G., L.R.C.P.&S.I., D.P.H., D.T.M.&H., Public Health Officer, Regional Office for Africa, World Health Organization.

GILLET, JOS. A., M.B., Ch.B., D.P.H., Medical Officer of Health and Chief School Medical Officer, County Borough of Rotherham.

HARDING, W. G., M.R.C.S., L.R.C.P., D.P.H., Deputy Medical Officer of Health Metropolitan Borough of Wandsworth, and Assistant Divisional Medical Officer, Public Health Department, London County Council.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Divers.—On October 26, 1951, at the Redhill County Hospital, Surrey, to Betty (formerly Craig), wife of Dr. Lloyd Divers, a daughter.

Drummond.—On October 1, 1951, to Margaret, wife of James W. Drummond, M.B., Ch.B., Hatfield, Salisbury, Southern Rhodesia, a son.

Franklin.—On October 27, 1951, at the North Staffordshire Royal Infirmary, to Margaret (formerly McIlroy), wife of Dr. C. B. Franklin, a son.

Prestt.—On October 18, 1951, to June (formerly Coops), wife of Dr. John Prestt, of West Ridge Lodge, Oxton, Cheshire, a son—Jonathan.

Wolff.—On October 23, 1951, to Catherine Bernadette, wife of Dr. Frederick Wolff, a daughter.

MARRIAGES

Nash—Phillips.—On October 13, 1951, at Sidcup, Kent, Peter Howard Nash, M.D., of London, to Ada Elizabeth Phillips, of Adelaide, South Australia.

Old—Say.—On October 10, 1951, at Combe Down, Bath, Dr. Rowton Old to Pamela Say.

DEATHS

Bidie.—On October 30, 1951, at Edinburgh, George Bidie, M.D., F.R.C.S.Ed., Lieutenant-Colonel, I.M.S., retired.

Hadfield.—On October 27, 1951, at "Overhayes," Marazion, Cornwall, Rowland Hurst Hadfield, M.R.C.S., L.R.C.P., aged 71.

Lowe.—On September 2, 1951, at his home, 50, Merton Road, Harrow, Middlesex, Peter Lowe, M.B., Ch.B., D.P.H., formerly of Hereford, aged 77.

Mackenna.—On November 1, 1951, at Mellish Hotel, Mellish Road, Walsall, Staffs, David Lockhart MacKenna, M.B., Ch.B.

Moore.—On October 28, 1951, at 24, Minster Yard, Lincoln, Jane Kerry McElroy Moore, J.P., M.B., Ch.B.

Prentice.—On October 29, 1951, at 63, Cambridge Road, Southport, Alexander Prentice, M.B., C.M.

Robson.—On November 2, 1951, at King Edward VII Hospital, Windsor, William Gordon Robson, M.B., Ch.B., M.R.C.P., of Green Alley, Farnham Common, Bucks.

Scott.—On October 30, 1951, at Bolney, Blandford, Dorset, Leonard Bodley Scott, M.D., D.P.H., Lieutenant Colonel, I.M.S., retired.

Steen.—On October 29, 1951, at "Dunboe," Shepperton-on-Thames, Middlesex, Hugh Barkley Steen, M.D., M.Ch., Lieutenant-Colonel, I.M.S., retired.

Thomas.—On October 30, 1951, at 49, Victoria Road North, Southsea, Hants, Arthur Richard Thomas, O.B.E., F.R.C.S.Ed., Surgeon Rear-Admiral, R.N., retired, aged 75.

Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Early Ambulation

Q.—*How early should patients be got on their feet after abdominal operations such as appendicectomy or cholecystectomy? What is gained by early ambulation, and what are the risks? Do follow-up studies support the claims made for early ambulation?*

A.—It is the current practice of most abdominal surgeons to get their patients out of bed fairly soon after operation. Not unnaturally, there are considerable individual variations in this practice, but there is a general tendency to get patients up and about within two to four days of an abdominal operation. Some have suggested that it is important to have the patient walking within 24 hours, but most surgeons prefer a more considerate regime.

There is no doubt at all that most patients benefit greatly both in their morale and in their sense of physical well-being. Other advantages are: first, that urinals and bedpans are used to the very minimum, to the mutual advantage of nurses and patients; secondly, that there is a diminished incidence of post-operative pulmonary collapse (although it is stressed that early ambulation is merely one aspect of the modern "shake-up" regime); and, thirdly, there is probably a lower incidence of venous thrombosis. There seem to be no real risks associated with the intelligent practice of early ambulation. Anxiety about integrity of the suture line has proved unfounded. Some believe that wounds heal better, and there is experimental support for this view; at all events there is certainly neither a higher incidence of wound disruption nor a greater number of recurrences following hernia operations. Early rising should not, however, be used as a pretext for too early discharge from hospital care.

Follow-up studies do support the *reasonable* claims for early ambulation; they do not support extravagant claims that, for example, post-operative venous thrombosis is reduced tenfold in its incidence.

All these points are fully discussed with a good bibliography in the *British Medical Journal* (1948, **2**, 1026), and the questioner is referred to this leading article for a compact summary of the recent literature on this subject.

Excision of Patella after Fracture

Q.—*What are the indications for excising the patella after fracture? What is the prognosis? What exercises are suggested for rehabilitation after the operation?*

A.—Excision of the patella is advisable for comminuted fractures with displacement at all ages, and for transverse fractures with displacement in patients over the age of 45 years. The prognosis is good provided the operation is performed skilfully and is followed by an adequate period of intensive physiotherapy. The only common residual disability in such cases is slight impairment of power of extension of the knee, noticed when climbing stairs or ladders, and perhaps slight limitation of the range of flexion.

Exercises should be begun one week after operation. At first they should consist simply of active quadriceps contractions. After about three weeks, when the tendon repair is becoming sound, straight-leg-raising exercises and more intensive static quadriceps contractions are practised. As strength improves, resistance exercises are added. The object should be to secure a powerful quadriceps muscle, able to sustain the knee in full extension when raised from the bed, before flexion exercises are begun. In a favour-

able case it should be permissible to begin knee-flexion exercises between four and six weeks after operation. Thenceforth general knee-strengthening exercises are continued until function approaches normal. The patient usually gets up about four weeks after operation.

Prognosis in Infantile Eczema

Q.—*What is the prognosis in infantile eczema? Does it ever persist into childhood, or must an alternative diagnosis be considered? Is it usual for the eruption to become worse with excitement or anxiety?*

A.—For a variety of reasons, inborn or environmental, a sensitive infant may itch. Rubbing and scratching produce infantile eczema. The habit often ceases when the child starts walking or is past the phase of teething. In some it may persist for various reasons, psychological factors taking a high place. Prognosis therefore depends on circumstances. The family history in this regard may be a guide. Any structural abnormality, such as xeroderma, carries with it a less favourable prognosis.

If the affection persists it may be given other labels—such as Besnier's prurigo, constitutional eczema, or neurodermatitis—but the diagnosis is the same.

The affection invariably worsens with excitement or anxiety.

Osteochondritis Dissecans or Osteochondromatosis

Q.—*A man of 24 with pain in the left hip, particularly on walking after resting for a while, and limited and painful adduction, has been found on radiography to have several loose bodies in the joint, with others forming from the acetabulum. Is this osteochondritis dissecans of the hip?*

A.—Osteochondritis dissecans of the hip-joint, though rare, is a well-recognized condition, and there is usually a single lesion involving the upper surface of the femoral head. A good description is to be found in a paper by King and Richards (*J. Bone Jt Surg.*, 1940, **22**, 327). However, the patient described has several loose bodies in the hip-joint, and it is therefore more likely that he has osteochondromatosis, a benign neoplastic condition of the synovial membrane characterized by the formation of cartilaginous tumours which may ultimately be numbered by the hundred—see Kienböck, R. (1934), *Röntgendiagnostik der Knocken- und Gelenkkrankheiten*, vol. 3; Brailsford, J. F. (1948), *Radiology of Bones and Joints*, 4th ed., p. 308; and Shanks, S. C., and Kerley, P. (1950), *Textbook of X-ray Diagnosis*, 2nd ed., vol. 4, pp. 131 *et seq.* Many of these little tumours may be uncalcified and are then only revealed by contrast arthrography, see Bloom, R., and Pattinson, J. N. (1951), *J. Bone Jt Surg.*, **33B**, 80. The appropriate treatment of osteochondromatosis—total synovectomy—is technically difficult.

Conception without Penetration

Q.—*Is anything known about the powers of spermatozoa to penetrate clothing? A woman alleges that a certain man is the father of her child, but agrees with him in saying that ejaculation occurred on one occasion only, and that then she was wearing her knickers. Could conception occur under such circumstances?*

A.—There appears to be no definite information about the power of spermatozoa to penetrate clothing. However, in the circumstances described in the question it is quite possible that spermatozoa could have reached the introitus. Whether or no they could then have proceeded to the cervix and thence to the Fallopian tubes is quite another matter. Although it is true that a few rare authenticated cases have been recorded in which conception has occurred with an intact (though of course not imperforate) hymen, the overwhelming experience of those concerned with problems of fertility is that the survival of spermatozoa in the vagina is very limited, and the prospects of conception occurring, except where ejaculation takes place close to the external os of the cervical canal, are very small indeed. In short, therefore, although it is possible that conception might have occurred in the circumstances described in this question, the chances are overwhelmingly against it.

Subcutaneous or Intramuscular?

Q.—*Why are intramuscular injections preferred to subcutaneous in so many cases? Nurses are taught to give all injections except insulin into a muscle, and a well-known firm recommends the administration of their vaccines and toxoids intramuscularly. And yet subcutaneous injections lead to slower absorption (an advantage when antibiotics are given), less pain on movement, and less risk of local palsy should the recipient become infected with the virus of poliomyelitis.*

A.—It is not the general practice for nurses to be taught to give *all* injections except insulin intramuscularly. Certain drugs cause pain and sloughing when given subcutaneously, and must be given intramuscularly. Further, if a large volume has to be injected, the intramuscular route is much preferable. There are numerous preparations, however, to which these objections do not apply.

Post-prandial Leucocytosis

Q.—*Is it true that certain types of food—e.g., tinned foods and foods which have been pressure-cooked—produce a post-prandial leucocytosis while others do not?*

A.—It is not generally agreed by haematologists that food of any kind produces leucocytosis, and there is no evidence available suggesting that certain types of food produce this effect more than others. A small increase in leucocytes has, however, been observed one hour after a protein meal, particularly if there has been a previous period of fasting; the effect is maximal after four hours and subsides entirely after six hours. It is well known that an appreciable rise in leucocytes occurs in all persons during the afternoon, and it is this "afternoon tide," as it is called, which is often confused with post-prandial leucocytosis.

NOTES AND COMMENTS

Preservatives and the E.S.R.—Mr. T. D. WHITTET, Chief Pharmacist at University College Hospital, London, writes: I noticed that you have had an inquiry (October 27, p. 1040) about the effect of preservative on sodium citrate solution used in the E.S.R. estimation. An interesting paper on this subject, by H. Henriksen, appeared in the *Pharmaceutical Journal* (1942, **148**, 14). In a series of controlled experiments the author showed that the use of 0.1% chlorocresol did produce errors outside the limit of experimental error of the method used (Westergren). He also showed that 0.002% of phenylmercuric nitrate (the other preservative recommended by the *British Pharmacopoeia*) did not interfere significantly with the test.

All communications with regard to editorial business should be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1. TELEPHONE: EUSTON 4499. TELEGRAMS: *Aitiology, Westcent, London.* ORIGINAL ARTICLES AND LETTERS forwarded for publication are understood to be offered to the *British Medical Journal* alone unless the contrary be stated.

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